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VOLUME 42 NUMBER 2

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AUGUST 1945

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO 10, ILLINOIS. ANNUAL SUBSCRIPTION, $6.00

Entered as Second Class Matter Jan. 14, 1925, at the Postoffice at Chicago, Under the Act of Congress of March 3, 1879

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OTOLOGIC RESEARCH IN BRITAIN

NOTES ON ITS PROGRESS

C. S. HALLPIKE, F.R.C.P., F.R.C.S.

Member of the Scientific Staff of Britain's Medical Research Council; Aural Physician and Director of the Otological Research Unit, National Hospital, Queen Square, London.

LONDON, ENGLAND

Research in otology has always been characterized by a high degree of specialization, particularly in the fields of anatomy and pathology, and well before the end of World War I the claim that special provisions were needed to meet its requirements had received wide support. This claim attained recognition in the establishment of special centers, such as the otologic laboratory at the Johns Hopkins Hospital, Baltimore, and the Ferens Institute at the Middlesex Hospital, London, and has since been abundantly justified by the results achieved.

During the present war, structural damage has compelled the closing of the Ferens Institute, and otologic research in Britain has had of necessity to confine itself in the main to problems directly connected with the needs of the fighting forces.

The administrative control of clinical material which is possible under service conditions exists in some ways the control attainable in civilian life, an advantage which has been exploited with particular energy by the Central Medical Establishment of the Royal Air Force. Investigations of the wide range of otologic problems which arise in flying personnel have been systematically pursued by the ear, nose and throat department of the establishment, and some of the results obtained promise to have applications extending well beyond the sphere of service conditions.

Vital Statistics.—The want of adequate morbidity data relating to deafness and chronic disease of the ears has long been in urgent need of remedy. It seems likely that valuable information of this kind will be provided by the detailed analyses which have been made by the ear, nose and throat specialists of the R. A. F. of aural conditions causing rejections at air crew medical boards. It will be of particular interest to compare these data with the results of Beasley's masterly survey of aural defects encountered in a sample of the American urban population.

Vacuum Otitis or Barotrauma.—In the years immediately preceding World War II, this serious disabilty of aviators was a rarity among the highly selected flying personnel of the R. A. F., and, with the subsequent huge expansion of the force, there seemed no reason to expect that it would become one of the major medical causes of operational inefficiency. Yet this has proved to be the case.

The work of Wing Commander McGibbon and others on the mechanism of this condition and its pathologic anatomy, as reproduced in cats, has done a great deal to stimulate interest in the difficult subject of the eustachian tube, and it seems safe to predict that certain of the methods which have been elaborated for the testing of tubal efficiency, particularly the systematic use of the decompression chamber, will come to have a wide application in the study of tubal obstruction as it occurs in the civilian population.

Tests of Hearing.—The difficulties which attend the use of the whispered voice as a regulation test of the hearing of those who enter the fighting forces are well known and various—in particular, the difficulty of accurate standardization. For this reason, much attention has been devoted to the possibility of its replacement by pure tone audiometry.

Various lines of evidence—and mention should here be made of the work of Fowler and of Steinberg—have for some time conveyed a clear suggestion that pure tone audiometry tests an aspect of hearing which is related in no simple way to that involved under operational conditions in aircraft, with strong speech signals received against a background of high level ambient noise.

Further confirmation of this point has been provided by the results of a searching inquiry conducted by the staff of the acoustics laboratory of the R. A. F. Central Medical Establishment.

Tests were carried out on air crew personnel with pure tone audiometry in quiet conditions and also with word articulation in conditions of artificial aircraft noise, and a comparison of the results showed an interesting lack of correlation.

Factors Affecting the Efficiency of Intercommunication Equipment in Noise.—These factors have been extensively studied by the Royal Aircraft Establishment in connection with the intercommunication equipment in aircraft, while a high measure of success has been achieved by an expert committee of Britain's Ministry of Supply in the designing of communications equipment which gives high articulation efficiency under the extremely adverse noise conditions of certain kinds of tanks.

This work makes a particular claim on the attention of the otologist, since the problems involved are in many ways akin to those which attend the designing of hearing aids for the deaf.

In this connection, it is a matter of unusual interest that the particular characteristics of the moving coil microphones and receivers, which were found to determine the superiority of this kind of equipment at the high noise level encountered in armored fighting vehicles—i. e., smoothness of frequency response and freedom from distortion at a high level of signal strength —were also noted by Littler in 1936 as being responsible for its superiority when used as hearing aids for the deaf.

The Medical Research Council of Great Britain and Otologic Research.—From the viewpoint of state medicine, sickness and mortal illness would appear to be the natural counterparts of wounds and deaths on the battlefield. While it is a commonplace that military problems relating to the wounded man on the battlefield far exceed those relating to the dead and have commanded a correspondingly greater attention on the part of military medical authorities, it cannot yet be said that sickness rather than mortal illness has dominated the attention of civil health authorities to the same extent. Nevertheless, the trend of vital statistics shows an increasing concern with disability rather than with death, with morbidity data rather than with death rates; and this trend is particularly pertinent to a source of disability so widespread and intractable as deafness and chronic ear disease.

This realization of the national importa disease of the ear has recently found expres in the establishment by the Medical Res Council of a number of committees to furt wide program of research into the proble deafness and its causes.

One of these committees will deal with medical and surgical problems of deafnes chairman is Prof. Henry Cohen, of the fa of Medicine, Liverpool University, and its s tary, Mr. T. E. Cawthorne, aural surgeo Kings College Hospital, London.

Problems connected with the education o deaf will be dealt with by a second comm under the chairmanship of Prof. F. C. Bar The secretary of the committee is Dr. A. W Ewing, director of the department of educ of the deaf at Manchester University.

A third committee has been appointed, to with problems of design and performanc electroacoustic equipment used in the inves tion and alleviation of deafness, includin matters connected with audiometers and el hearing aids. The chairman is Dr. W. G. ley, controller of research, post office engine department, and the secretary. Dr. T. S. Li lecturer in acoustics, Manchester Univer Through the work of this committee, it is h that much valuable experience gained i designing of electroacoustic equipment to the needs of the fighting forces during the few years of war will now be turned to advantage of the deaf.

In addition, the Medical Research Co jointly with the National Hospital for Ner Diseases, Queen Square, London, has rec established at the hospital an otologic res unit the work of which is to be so directed a take advantage of the wealth of clinical ma available at the hospital and thus to advanc search in the numerous and important prob of otoneurology. Apart from the clinical com ments of this unit, it is anticipated that laboratory accommodation at its disposal make possible the maintenance of adequate fa ties for work in electrophysiology, histolog the temporal bone and other important of otologic research.

LABYRINTHITIS FOLLOWING PURULENT INFECTION OF THE MIDDLE EAR

HISTOPATHOLOGIC STUDIES

FRANZ ALTMANN, M.D.
AND
JULES G. WALTNER, M.D.
NEW YORK

e recent advances in the chemotherapy of ent infections have radically changed the de of physicians toward the problem of nogenic labyrinthitis. In the past their concern had been the prevention and the ment of meningitis, which often followed inthitis and which very often had a fatal nation. Today, however, meningitis has e somewhat less a problem. The main in- has shifted to the question of the preven- nd the treatment of labyrinthitis. The pre- n of labyrinthitis means the preservation of ng, which is greatly endangered and too fre- ly lost in cases of labyrinthine infection. A ugh knowledge of the genesis of this disease , therefore, highly desirable.

a previous paper [1] the genesis of labyrin- following subacute otitis media due to Pneu- ccus type III was discussed. In this paper inthitis following exudative inflammation of middle ear will be studied and particularly le played by the windows in the genesis of byrinthine changes analyzed. A review of terature shows that this role is not always y understood and that in many instances the lexity of the problems arising in this connec- s not clearly realized.

these circumstances it seemed worth while port and discuss the observations in 5 repre- tive cases.

o of the cases were observed in the Presby- Hospital in New York, 1 in Lenox Hill ital in New York and 2 in the General Hos- in Vienna, Austria.

e authors were assisted by grants from the rch Council of the American Otological Society e Hayden-Coakley Fund.

m the Department of Otolaryngology of the bia University College of Physicians and Sur- and the Presbyterian Hosptial.

Altmann, F., and Waltner, J. G.: Labyrinthitis Pneumococcus Type III, Arch. Otolaryng. **40:** ug.) 1944.

REPORT OF CASES

CASE 1.—G. S., 2 years of age, was admitted to the General Hospital in Vienna on June 25, 1927. Three weeks before this, in the course of an infection of the upper respiratory tract, she had a retroauricular swelling which lasted for a few days.

The external auditory canal was dry. There was beginning sagging of the posterosuperior canal wall. The drum membrane was pale red, with no landmarks visible. The right drum membrane was normal. The nose and throat showed no pathologic changes.

The retroauricular swelling was incised and a large subperiosteal abscess opened which led into a cavity in the mastoid process. At the bottom of the cavity the lateral sinus was found exposed. Culture of the pus showed hemolytic streptococci. Two days later, because of a chill and a rise in temperature to 104 F., the lateral sinus was further exposed and incised and a thrombus removed. Culture of the blood showed hemolytic streptococci. Two days after the second operation, meningeal signs developed. The spinal fluid was cloudy and contained hemolytic streptococci. Four days later the child died.

Labyrinthine signs were not noticed at any time.

Autopsy showed acute cerebrospinal leptomeningitis caused by hemolytic streptococci.

Histologic Examination of the Left Temporal Bone (vertical serial sections).—(*a*) Middle Ear: The lumen contained a moderate amount of purulent exudate. The cavity was lined with hyperplastic mucosa with acute inflammatory changes in its superficial layers. The epitympanum and the window niches contained masses of organization tissue with cyst formation (fig. 1). The ossicles were embedded in masses of hyperplastic mucosa. The acute inflammatory infiltration was more marked in the mucosa of the window niches than in the rest of the mucosa. The area in which the membrane of the round window normally is attached to the medial surface of the promontory was occupied by a large abscess, and the membrane was detached from the bone. The abscess cavity communicated freely with the scala tympani, and both contained fibrinopurulent exudate with a considerable amount of blood. The abscess extended outward in the deep layers of the mucosa along the inner surface of the promontory almost to its convex lateral surface. The round window membrane was heavily infiltrated in the neighborhood of the abscess, and the infiltration decreased gradually in intensity toward the rest of the membrane.

The eustachian tube contained some pink-staining exudate. Its mucosa was free from acute inflammatory changes.

Pneumatization was confined to cells in the tegmen tympani and in the adjacent parts of the squama and to

a few perilabyrinthine and retrolabyrinthine cells. The cells were lined with hyperplastic mucosa and contained chronic inflammatory exudate with small and large round cells. Acute inflammatory changes were not present in the cells.

The jugular bulb was free from inflammatory changes.

(*b*) Inner Ear: The cochlea contained hemorrhagicopurulent exudate. Corti's organ and the adjoining structures were almost completely destroyed. Reissner's membrane was entirely missing. The exudate extended into the innermost part of the cochlear aqueduct. The vascular and nerve channels of the modiolus contained purulent exudate. Many cells of the spiral ganglion were destroyed, and others were degenerating.

The vestibule contained purulent exudate and the necrotic remnants of the utricle and the saccule. The vestibular surface of the footplate of the stapes was denuded of endosteum and covered with pus. The inner parts of the annular ligament were destroyed; the outer parts, infiltrated with leukocytes. The leukocytic infiltration extended into the mucosal covering of the footplate. The latter in its anterosuperior part was slightly dislocated toward the middle ear. The endolymphatic duct and sac contained purulent exudate.

In the semicircular canals, the perilymphatic and endolymphatic spaces were filled with purulent exudate, and the membranous parts were completely necrotic. The vascular and nerve channels leading into the internal auditory meatus, particularly the channel for the posterior ampullar nerve, were infiltrated with leukocytes.

The arachnoidal cisterna in the fundus of the in auditory meatus was filled with pus, and there w interstitial purulent infiltration of the fibers of the nerve. The facial nerve was free of changes. medial part of the internal auditory meatus con free pus. The periosteum was free from inflamm changes.

Summary.—In a 2 year old girl a retroauricular periosteal abscess developed three weeks after an tion of the upper respiratory tract. Simple mast tomy was performed and a perisinuous abscess dr Two days later the sigmoid sinus was opened a thrombus removed. The patient died four days the second operation, from a labyrinthogenic pu cerebrospinal meningitis. As often in young chi labyrinthine symptoms were not noticed, and the

Fig. 1 (case 1).—The stapes is embedded in cystic tissue and slightly dislocated toward the middle ear. lateral part of the membrane of the round window is destroyed by an abscess which has perforated into the tympani. The vestibule contains purulent-necrotic exudate.

time of the onset of the labyrinthine infection cou be determined.

Histologic examination showed the tympanic c lined with hyperplastic mucosa which presented inflammatory changes, most marked in the w niches. The round window membrane had been de from its insertion point at the inner surface of the montory by a large abscess in the deep layers o mucosa. This abscess had broken into the scala ty and caused diffuse purulent-necrotizing labyrinthitis spread of the infection of the internal auditory meatu from there to the leptomeninges. There were l cytic infiltration of the outer parts and destructi the inner parts of the annular ligament, with outward dislocation of the footplate of the stapes.

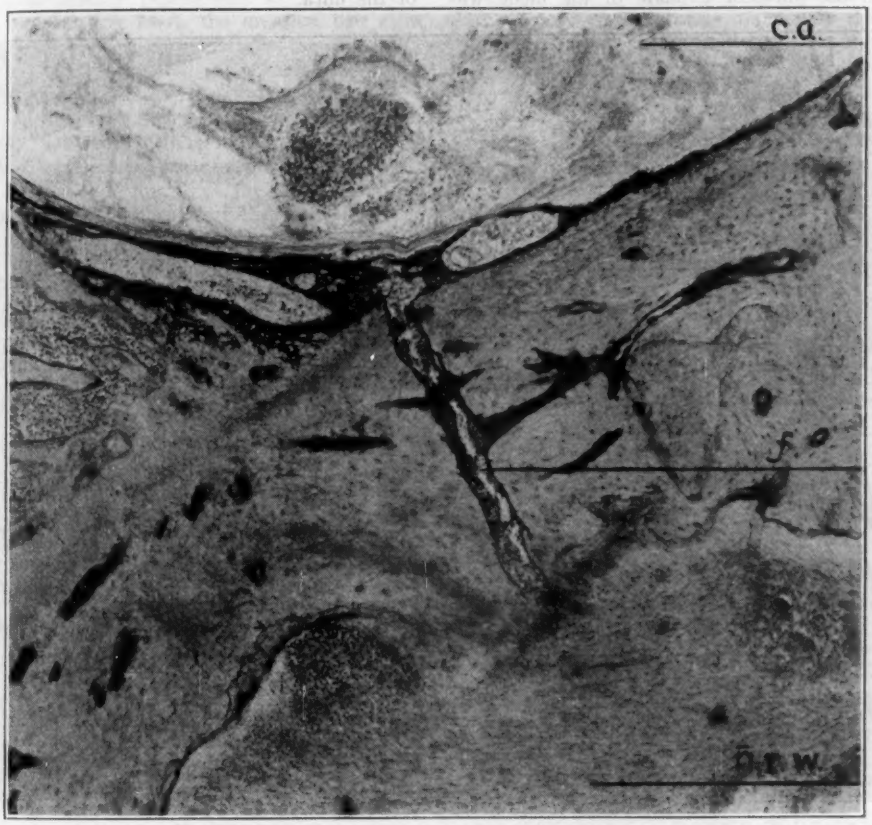
es in the oval window were much less marked those in the round window. They were apparently equela and not the cause of the labyrinthine ion.

E 2.—F. N., 46 years of age, was admitted to Hill Hospital on April 29, 1937. He had noted ic intermittent purulent discharge from the left r fifteen years. In the first days of March 1937 he e dizzy and had to stay in bed for a few days. ar started to discharge again, and the dizziness, a short intermission, became worse. The patient dmitted to the hospital on March 27. At that time were conduction deafness in the left ear averaging cibels of loss and a slightly hyperactive response to c stimulation of the left labyrinth. Under con-

cocci. Two days later the patient died, fifteen days after the onset of the labyrinthine symptoms.

Autopsy showed acute purulent cerebrospinal leptomeningitis due to the hemolytic streptococcus.

Histologic Examination of the Left Temporal Bone (horizontal serial sections).—(*a*) Middle Ear and Mastoid Process: There was a large radical mastoidectomy cavity. The facial ridge was low, and the incus and the malleus had been removed. The mucosa was preserved only in the window niches. It was thick, hyperemic, edematous, and infiltrated with polymorphonuclear leukocytes. The mastoid process consisted of diploic bone with increasing sclerosis toward the periphery. A few periantral cells were left and were lined with thick, infiltrated mucosa.



ig. 2 (case 2).—Purulent infiltration of the fissure between the niche of the round window and the ampulla e posterior vertical canal. *c.a.* indicates the crista ampullaris of posterior vertical canal; *f.*, the fissure; *n.r.w.*, iche of the round window.

tive treatment, the condition of the patient improved, he dizziness disappeared. On April 25, four days e his second admission, he felt shooting pain in the ar, increase in the secretion, considerable reduction aring and severe dizziness. The symptoms per-, and the patient was readmitted to the hospital.

e drum membrane showed a large perforation, and mucosa of the middle ear appeared to be necrotic. May 4, radical mastoidectomy was performed. mastoid process was sclerotic and the middle ear with granulations. The hammer and the incus removed. The stapes seemed to be a little loose. e was no evidence of cholesteatoma.

ur days after the operation, the patient became sy, and the temperature rose to 105.8 F. The l fluid was cloudy and contained hemolytic strepto-

The petrosal tip consisted of diploic bone. Fibrosis of the fat marrow and limited new formation of bone were seen in some of the marrow spaces. The tympanic ostium of the eustachian tube was closed by a plug consisting of leukocytes, fibrin and red blood cells. The niche of the round window contained fibrinopurulent exudate. The membrane of the round window showed near its insertion point at the inner surface of the promontory a leukocytic infiltration throughout its entire thickness and a similar but less marked infiltration near the opposite insertion point. The rest of the membrane showed a slight infiltration of its superficial layers but was otherwise intact. The fissure between the niche of the round window and the ampulla of the posterior vertical canal was well developed and heavily infiltrated with leukocytes (fig. 2). The niche of the

oval window contained fibrinopurulent exudate (fig. 3). Its anterior part was covered with fibrinous masses, leukocytes, red blood cells and some small bone splinters. The stapes seemed to be rotated posteriorly around a vertical axis. The posterior part of the footplate protruded somewhat into the vestibule and the anterior part into the middle ear. The cartilaginous lining of the footplate and of the window frame was intact. The annular ligament was missing with the exception of a few small remnants which were attached to the posterior part of the footplate. Only in the anterior part of the footplate was the mucosal covering still in direct continuity with the mucosa of the window niche, helping to keep the stapes in place. In the other parts of the circumference this connection had been lost. The middle ear communicated with the vestibule, and the purulent exudate of the niche was exudate, which also extended into the innermo of the aqueductus cochleae. Reissner's membra Corti's organ were completely destroyed. The spiralis secundaria and the adjoining part of the membrane were partially destroyed, and an ab communication had been established between th end of the scala tympani and the vestibule. The a diffuse purulent infiltration of Rosenthal's ca of the vascular and nerve channels of the modio

In the vestibule and semicircular canals, the the saccule and the membranous canals, includi nerve endings, were almost completely necrotic perilymphatic spaces contained purulent exudate endolymphatic duct and sac were well preserv filled with pus. The perisaccular tissue was infi with leukocytes, but there was no actual pene of the dura.

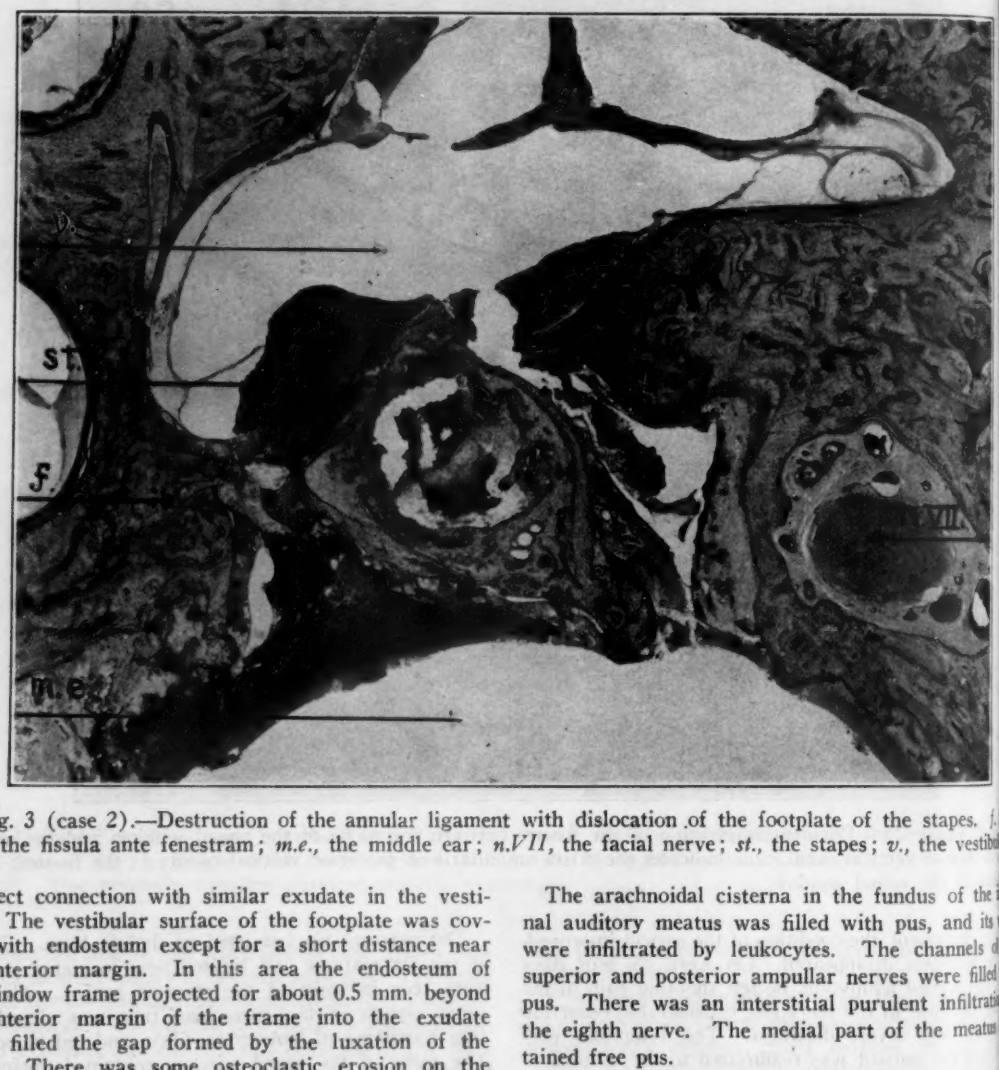


Fig. 3 (case 2).—Destruction of the annular ligament with dislocation of the footplate of the stapes. *f.* cates the fissula ante fenestram; *m.e.*, the middle ear; *n.VII*, the facial nerve; *st.*, the stapes; *v.*, the vestibu

in direct connection with similar exudate in the vestibule. The vestibular surface of the footplate was covered with endosteum except for a short distance near the anterior margin. In this area the endosteum of the window frame projected for about 0.5 mm. beyond the anterior margin of the frame into the exudate which filled the gap formed by the luxation of the stapes. There was some osteoclastic erosion on the surface of the capitulum. The tendon of the stapedial muscle, including its insertion point, was intact. The mucosa in the neighborhood of the tympanic end of the fissula ante fenestram was densely infiltrated with polymorphonuclear leukocytes (fig. 3). From here the infiltration extended into the connective tissue which formed the central part of the fissula and throughout the entire length to its vestibular end.

(*b*) Inner Ear: In the cochlea, the scala tympani and the scala vestibuli were filled with fibrinopurulent

The arachnoidal cisterna in the fundus of the i nal auditory meatus was filled with pus, and its were infiltrated by leukocytes. The channels superior and posterior ampullar nerves were filled pus. There was an interstitial purulent infiltrati the eighth nerve. The medial part of the meatu tained free pus.

Summary.—A 46 year old patient had chronic mittent left-sided aural discharge for fifteen During an acute exacerbation of the suppur dizziness developed and lasted for about four The labyrinthine function was still preserved a time. Three weeks later the patient had another of dizziness with rapid loss of hearing. A ra mastoidectomy was performed, and the stapes app somewhat loose. Four days after the operation,

after the onset of the labyrinthitis, the patient
of purulent meningitis.
tological examination of the temporal bone showed
ction of the annular ligament with dislocation
e footplate of the stapes and diffuse purulent-
izing labyrinthitis. The peripheral parts of the
window membrane, particularly in its lateral
were heavily infiltrated with leukocytes. From
byrinth the infection had spread mainly through
erve and vascular channels of the modiolus into
rachnoidal cisterna in the fundus of the internal
ory meatus and from there to the meninges, caus-
iffuse purulent meningitis.
e higher intensity of the changes in the oval
w indicated that the latter was in all probability
ite of the primary invasion of the labyrinth.
rding to the history (appearance of severe dizzi-
and marked hearing loss), the invasion had most
bly taken place nine days before the operation
had caused destruction of the annular ligament.
explains why the stapes was found loose during
peration. The large hole in the mucosal covering
e footplate and the projection of the endosteum
e window frame into the exudate suggest addi-
traumatism in the course of the operation.
e changes in the membrane of the round window
he purulent infiltration of the fissula ante fenes-
and of the fissure between the round window
and the posterior vertical canal were less ex-
ve and evidently of shorter duration than those
e oval window. They were, in all probability,
dary to the labyrinthine infection.

se 3.—J. K., 18 years of age, was admitted to
General Hospital in Vienna on Sept. 2, 1925.
tion had drained from the left ear for the past
eeks, following swimming. The night before ad-
on the patient suffered from a rise in temperature
headaches. The temperature on admission was
F. As a child he had had measles and mumps.
e right drum membrane was slightly thickened
retracted. The left drum membrane showed a
central perforation with profuse mucopurulent
arge. There was some sagging of the postero-
ior wall of the osseous canal. The tip of the
oid process was tender. There was a moderate
e of conduction deafness (conversational voice
heard at a distance of 18 inches [0.5 meter] when
ther ear was masked by means of Bárány's noise

modified radical mastoidectomy was performed.
mastoid process was well pneumatized, and the
were filled with pus and granulation tissue. The
oid sinus was exposed and a large perisinuous
ss emptied. Six days after the operation the
nt complained of dizziness. There was sponta-
horizontal rotatory nystagmus to the healthy
which was hardly increased by calorization with
water (67 F.) but changed its direction after
ization with hot water (110 F.). Owing to a
stent temperature around 102 F. which could not
plained by any other changes, the jugular vein was
nd the lateral sinus plugged off thirteen days after
rst operation. No thrombus was found.
ter the second operation the temperature remained
On September 27 the patient became drowsy
died the following day, twenty days after he had
complained of dizziness.
topsy showed diffuse purulent cerebrospinal lepto-
ngitis of long standing and pachymeningitis interna
e posterior surface of the left petrous pyramid.
culture showed hemolytic streptococci.

Histologic Examination of the Left Temporal Bone (vertical serial sections).—(*a*) Middle Ear: The mucosa presented the characteristics of marked hyperplasia with a moderate acute inflammatory infiltration of the superficial layers in some places. The attic and many other recesses were filled with cystic hyperplastic mucosa, which also surrounded the hammer. The incus had been removed at the first operation. The round window niche contained cystic mucosa, the deep layers of which were transformed into granulation tissue, and this had grown through the window into the lowest part of the scala tympani and from there into the innermost part of the aqueductus cochleae. The membrane of the round window was to a large extent destroyed, but the osseous frame was intact. The stapes was covered with hyperplastic mucosa and considerably dislocated toward the middle ear (fig. 4). The annular ligament was completely destroyed. The mucosa which covered the stapes had remained everywhere in contact with the neighboring mucosa, and the middle ear was still completely separated from the vestibule. The footplate and the anterior crus of the stapes were partially necrotic, and active erosion by osteoclasts was seen. The deep layers of the mucosa in the vicinity of the oval window had been transformed into granulation tissue which covered the entire circumference of the window frame in a thick layer. The window had been markedly widened by vivid osteoclastic erosion from the surface of the bone. The areas of the promontory adjacent to the oval window showed a marked degree of rarefying osteitis.

The eustachian tube revealed moderate catarrhal inflammation of the mucosa.

(*b*) Mastoid Process: Many perilabyrinthine cells remained unopened. Well developed cells below, above and behind the labyrinth were lined with hyperplastic mucosa; only a few of them showed a purulent infiltration of the mucosa and pus in the lumen. In the deep cells of the petrosal angle, immediately behind the labyrinth, an abscess the size of a bean had been formed. This abscess communicated through a narrow fistulous tract with the operative cavity. It had eroded the internal cortical layer and produced an epidural abscess of the posterior fossa.

Another, larger, epidural abscess of the posterior fossa had been formed from diseased retrolabyrinthine cells. The covering dura was heavily infiltrated with polymorphonuclear leukocytes. The labyrinthine capsule showed an area of marked rarefying osteitis in the neighborhood of these diseased groups of cells.

(*c*) Inner Ear: The lumen of the cochlea contained purulent exudate and debris. The upper part of the middle turn and the lower part of the apical turn were filled with organization tissue, in which the lamina spiralis ossea, the basilar membrane and some remnants of the organ of Corti were still visible (fig. 5). In the rest of the cochlea the previously mentioned structures were completely destroyed with the exception of a few remnants of the lamina spiralis ossea in the basal turn. Rosenthal's canal in the middle cochlear turn showed proliferation of the connective tissue elements between the ganglionic cells. In the other parts, there was only a moderate infiltration of the vascular and nerve channels, and the cells of the spiral ganglion were well preserved.

The lumen of the vestibule and semicircular canals was filled with pus. The utricle, the saccule and the membranous canals were necrotic.

The endolymphatic duct and sac were filled with pus. A large perisaccular abscess had been formed in the dura of the posterior fossa, and the inner surface of

the dura in this region was covered with fibrinopurulent membranes.

In the internal auditory meatus, the sheath of the eighth nerve was infiltrated with round cells, and the arachnoidal cisterna in the fundus contained some small and large round cells but no polymorphonuclear leukocytes.

Summary.—In an 18 year old patient with a central perforation of the left drum membrane a reinfection of the middle ear developed after swimming. A modified radical mastoidectomy was performed because of high fever and headaches. Six days after the operation, signs of labyrinthine involvement were observed. The high fever persisted, but revision of the cavity and exploration of the sinus failed to give an explanation for it. The patient died of purulent meningitis twenty days after the first attack of dizziness.

changes were least marked in the upper part of the cochlea. The latter was filled with organization and contained remnants of the soft parts.

In addition to the perisaccular abscess, two epidural abscesses of the posterior fossa had formed from diseased retrolabyrinthine cells which not been drained. From all the three epidural abs a spread to the leptomeninges could be seen.

The changes in the oval window were more sive than those in the round window. This seem indicate that the changes in the former were the ones. The labyrinthitis was less intense in the part of the cochlea than in the rest of the labyr The two just mentioned facts favor the assum that the infection of the labyrinth occurred prim through the oval window and vestibule and not th the round window and scala tympani. Infection

Fig. 4 (case 3).—The annular ligament is destroyed and the stapes considerably dislocated toward the m ear. There is marked osteoclastic destruction of the window frame. The middle ear is completely separated the vestibule.

Histologic examination showed hyperplastic mucosa with only moderate acute inflammatory changes in both window niches. The deep layers of the mucosa were transformed into granulation tissue. The annular ligament of the stapes had been completely destroyed, the stapes had been dislocated toward the middle ear and granulation tissue growing toward the vestibule had caused marked osteoclastic erosion of the osseous frame of the window.

The round window membrane was to a large extent, but not completely, destroyed, and granulation tissue had grown into the basal part of the scala tympani.

There was diffuse purulent-necrotizing labyrinthitis with formation of a large perisaccular abscess. The

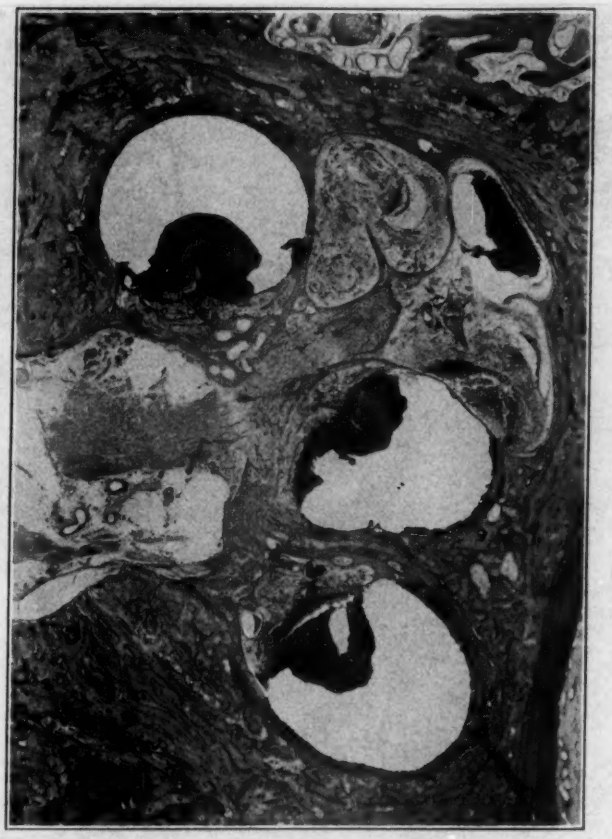
vancing simultaneously through both windows ca be ruled out completely but seems less probable infection by the oval window route.

Case 4.—E. B., aged 26, was admitted to the P byterian Hospital in New York, on April 29, (A more detailed clinical history of this case has given by Fowler and Swenson.[2]) One year previ a simple mastoidectomy had been performed on right side for acute purulent otitis media. Becau persistent aural discharge, right-sided headaches

2. Fowler, E. P., Jr., and Swenson, P. C.: rositis: A Roentgenologic and Pathologic Correl Am. J. Roentgenol. **41**:317, 1939, case xxiv.

eated pain behind the right eye, the patient had
one several subsequent operations, including a
mastoidectomy in October 1934. At that time
d an average hearing loss of about 40 decibels
r conduction in the speech range. In spite of
e operations, the symptoms persisted, and on
27, two days before his last admission, he had
ent attack of dizziness, after which he felt weak
roggy.
the day of admission the petrosal tip was ex-
by the Eagleton approach. The middle ear
was found completely filled with granulation
ar tissue, which was removed with a large curet.
ee pus was found in the tip.

lymphocytes and plasma cells, and was lined with pseudostratified columnar epithelium containing numerous goblet cells. In many places mucosal folds projected into the lumen. The latter contained a moderate amount of mucopurulent exudate. The mucosa of the eustachian tube was only moderately infiltrated with round cells. The posterior part of the operative cavity was lined with granulation tissue. The lumen was considerably narrowed because of formation of masses of scar tissue which filled out the hypotympanum and and a large part of the attic. In both areas there was extensive new formation of bone. In the hypotympanum the latter was deposited on the surface of the old bone. In the attic, the tegmen had been removed



ig. 5 (case 3).—The lumen of the cochlea contains exudate and debris. The upper part of the middle turn he lower part of the apical turn are filled with organization tissue.

e pain and the discharge persisted, and two fur-
attempts were made by approach from the middle
to explore the portion of the petrous pyramid
which the pus seemed to come, but without
ss.
July 6 signs of generalized meningitis developed,
he patient died five days later, seventy-four days
the violent attack of dizziness.
topsy showed acute fibrinopurulent cerebrospinal
ngitis caused by hemolytic streptococci and bilateral
ar pneumonia.

stologic Examination of the Right Temporal Bone (horizontal serial sections).—(*a*) Middle Ear: The
le ear formed the anterior part of the operative
y after the radical mastoidectomy. The mucosa
preserved in the anterior part and on the medial
It was thick, heavily infiltrated, mostly with

previously, and the newly formed bone was formed on the outer surface of the dura mater.

The malleus, the incus and the stapes were absent.

The niche of the round window contained a small amount of exudate. The mucosa which covered the round window membrane was thick and its deep layers heavily infiltrated with round cells, proliferated fibroblasts and polymorphonuclear leukocytes (fig. 6). It contained numerous thin-walled vessels which took a course perpendicular to the fibers of the membrane of this window. Some of the vessels and strands of the described cells could be followed inward through the membrane, particularly through its peripheral parts, into a mass of loose connective tissue containing many cells and thin-walled vessels. This tissue covered the inner surface of the round window membrane and filled out the lowest part of the scala tympani.

The defect in the lateral wall of the labyrinth caused by the absence of the stapes was bridged by a thick layer of mucosa, and thus the middle ear cavity was completely separated from the vestibule (fig. 7).

This mucosa was a continuation of that which covered the adjacent areas. In its deep layers it assumed the characteristics of granulation tissue. In the posterior half of the frame of the oval window the cartilaginous lining was still preserved. In the anterior half an extensive and still progressive destruction of the osseous frame and the adjacent parts of the promontory was taking place. The oval window had become considerably enlarged. The surface of the bony frame was covered with a thick layer of granulation

been opened during the operation, there was a of chronic osteomyelitis. The dura of the middle had been separated from the superior surface pyramid almost to the level of the gasserian ga In front of the cochlea there was an operative which extended from the superior to the po surface of the tip. The cavity was largely fil with newly formed bone, and the dura of both was covered with granulations.

(*d*) Inner Ear: The labyrinthine capsule in its periosteal layer marked rarefying osteiti ticularly in the neighborhood of the operative There were also numerous but smaller perivas resorption spaces within the enchondral layer.

Fig. 6 (case 4).—Granulation tissue growing into the lowest part of the scala tympani and from there int vestibule. *g.t.* indicates granulation tissue; *l.sp.o.*, the lamina spiralis ossea; *m.e.*, the middle ear; *n.r.w.*, the of the round window; *r.w.m.*, the round window membrane; *s.*, a bony sequestrum, a remnant of the dest lamina spiralis secundaria; *s.t.*, the scala tympani; *v.*, the vestibule.

tissue which protruded into the vestibule and contained several small bony sequestrums.

(*b*) Mastoid Process: The operative cavity was filled with scar tissue, in which extensive new formation of bone had taken place. The lumen was relatively small. A few perilabyrinthine cells were still present and were lined with thick and edematous mucosa. Some of them contained pink-staining homogeneous exudate; others contained disintegrating cells with cholesterol clefts between them.

(*c*) Petrous Tip: Pneumatic cells were completely missing. Behind and below the horizontal portion of the carotid artery, in a part of the tip which had not

The labyrinthine cavities contained masses of fib purulent exudate. The entire membranous labyr including the maculas and the cristas, was compl necrotic. The endolymphatic duct and sac with e tion of the part near the vestibule were free pathologic changes.

In the cochlea, the membrane of Reissner had completely destroyed, and the basilar membrane missing in some places. The contents of the mod were completely necrotic in the upper two turns the basal turn granulation tissue had grown from fundus of the internal auditory meatus along the cular and nerve channels into Rosenthal's canal

here into the adjacent parts of the scala tympani 8). The nerve fibers and ganglionic cells were ded in granulation tissue and gradually degen-. The previously described loose cellular con- tissue which filled out the lowest part of the ympani connected with the aforementioned tissue higher parts of the scala tympani of the basal In addition, it had penetrated through a breach separating structures into the adjacent part of estibule. The basilar membrane had been com- destroyed in this area, together with the inner of the lamina spiralis secundaria. The osteoclastic ction of the latter was still going on, and a few bony sequestrums had been formed, which were y pushed inward toward the vestibule by the re of the invading tissue (fig. 6).

showed an interstitial infiltration which rapidly decreased toward the central parts. The vestibular ganglion was markedly infiltrated, mainly by round cells and plasma cells. The ganglionic cells were degenerating. The facial nerve showed thickening and slight infiltration of its epineurium but was otherwise almost free from changes. The granulation tissue of the fundus extended into the facial canal almost up to the geniculate ganglion, and from there into the vestibule. It surrounded the nerve and caused marked osteoclastic destruction at the osseous walls of the canal, particularly toward the cochlea. Newly formed bone was seen in some places on the walls.

Histologic Examination of the Left Temporal Bone.—Beginning acute purulent infection of the middle ear

g. 7 (case 4).—Oval window. The stapes is completely missing, and the middle ear is separated from the oule by a thick layer of mucosa. There is extensive destruction of the anterior half of the window frame and djacent parts of the promontory. *m.* indicates mucosa separating the middle ear from the vestibule; *m.e.*, the le ear; *o.*, osteoclastic destruction of the anterior part of the window frame; *p.*, osteoclastic destruction of romontory; *v.*, the vestibule.

e arachnoidal cisterna in the fundus of the inter- uditory meatus was almost completely filled with lation tissue, which surrounded and invaded the hes of the cochlear and the vestibular nerve. In areas the nerve fibers had been completely de- ed. Accumulations of leukocytes were still visible me places within the granulation tissue. One of "residual abscesses" was situated in the central of the base of the cochlea; another one, in the where the arachnoidal septum joined the trunk e eighth nerve. In this region the arachnoidea nfiltrated with leukocytes and covered on its outer e with fibrinopurulent masses. The eighth nerve and the very first stages of meningogenic serofibrinous labyrinthitis were noted in the left temporal bone.

Summary.—On a 26 year old patient several operations, including eventually a radical mastoidectomy, had been performed because of persistent aural discharge, headaches and deep-seated pain behind the right eye. Two days after a violent attack of dizziness, the petrous tip was explored, but no free pus was found. Two further attempts at draining of the tip were made. The patient died of diffuse purulent leptomeningitis seventy-four days after the onset of the severe attack of dizziness, twenty-four days after the last operation.

Histologic examination showed diffuse purulent-necrotizing labyrinthitis of long standing, with granulation tissue growing into the labyrinthine cavities through the windows and from the fundus of the internal auditory meatus. The stapes was missing, and the oval window was filled with a thick layer of mucosa, the deep parts of which were transformed into granulation tissue which protruded into the vestibule. The round window membrane was still preserved but markedly infiltrated and its inner surface covered with young connective tissue. The fundus of the internal auditory meatus contained an abscess in a far progressed state of organization. The infection of the leptomeninges had taken place from an unorganized part of this abscess.

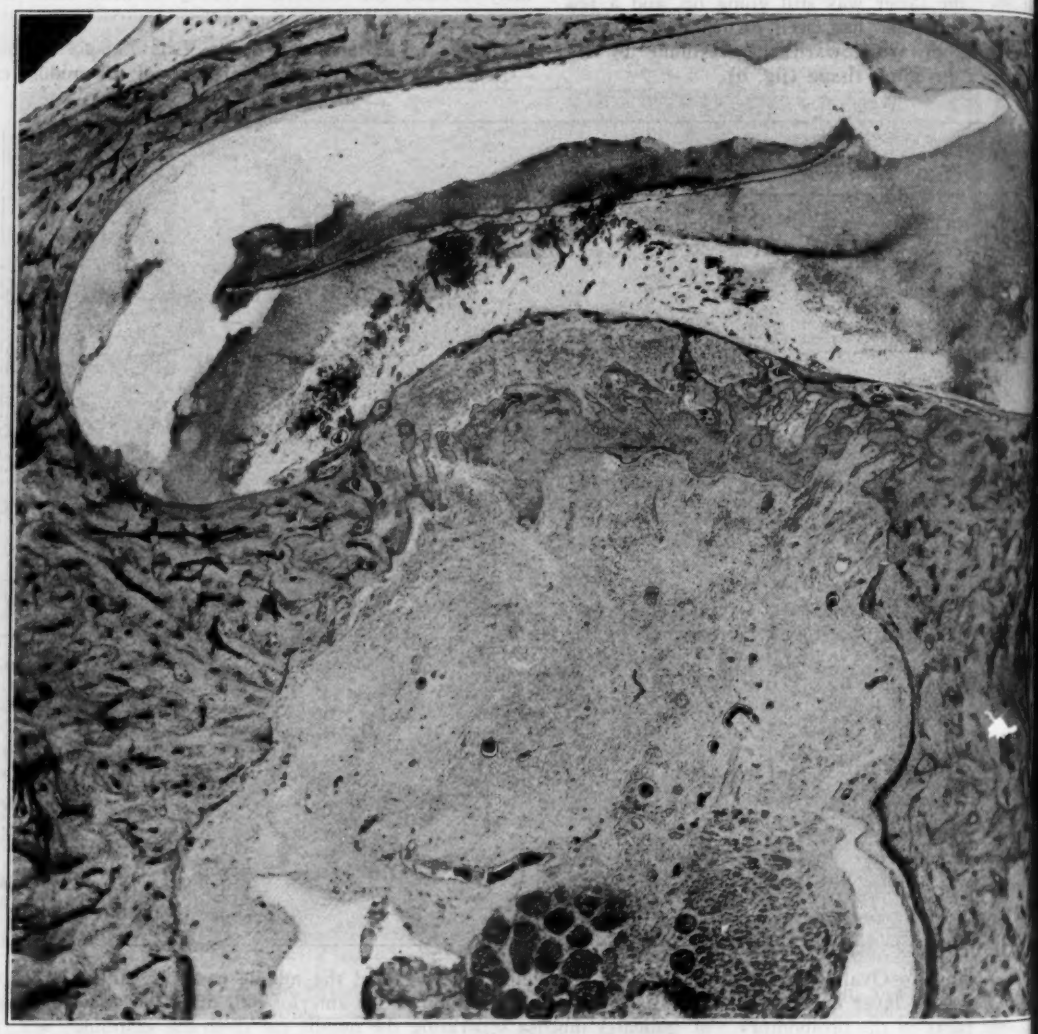


Fig. 8 (case 4).—Abscess of the fundus of the internal auditory meatus in a far progressed state of organization. Note granulation tissue growing into the basal turn of the cochlea.

The changes in the oval window were much more marked than those in the round window. There can hardly be any doubt that the former one represented the primary pathway of the labyrinthine infection.

CASE 5.—A 52 year old woman had a running left ear for forty years following scarlet fever. For the last eleven years she was under treatment for diabetes mellitus.

A radical mastoidectomy was performed on J... 1931. A fistulous opening was found in the re... the lateral semicircular canal. The hearing be... operation had been moderately reduced. After the... tion attacks of dizziness and nystagmus were ... for a few days, but these symptoms readily disa... on removal of the packing. Because of recurre... the dizzy spells, the operative cavity was revi... month later, but only sloughing soft tissue was ... in it. Three and one-half months later the patie... readmitted to the Presbyterian Hospital with p...

meningitis. A second revision of the cavity was ... The dural plate in Trautmann's triangle was ... necrotic and the dura covered with granulations. ... patient died four days after the last operation.

Autopsy showed diffuse purulent cerebrospinal ... meningitis due to Str. haemolyticus.

Histologic Examination of the Left Temporal ... (horizontal serial sections).—(*a*) Middle Ear: ... middle ear formed the anterior portion of the mast... tomy cavity. It was lined partially with remna... the thick and edematous mucosa and partially ... granulation tissue. The mucosa contained cystic ca...

y places. The anterior part of the middle ear e ostium of the eustachian tube contained a small eatoma. The incus and the malleus were missing, e stapes was embedded in cystic mucosa which eavily infiltrated with polymorphonuclear leuko- The cystic cavities were packed with pus cells. nular ligament of the stapes was preserved in the or part of its circumference only (fig. 9). The parts were either completely missing or in a far ssed stage of destruction. In these places, the nd middle thirds of the ligament were missing, the outer third was heavily infiltrated with leuko- The gaps which were formed by destruction of nular ligament were filled with granulation tissue grew inward into the granulation tissue within stibule and outward into the heavily infiltrated l covering of the footplate of the stapes. It walls. The operative cavity was lined with granulation tissue. The surface of the underlying bone was covered with a rather thick layer of newly formed bone in trabecular arrangement. The petrous tip consisted of diploic bone. Most of the marrow spaces contained fat marrow. The latter showed extensive fibrosis and round and plasma cell infiltration. New formation of bone was rather frequently seen in the marrow spaces, which in some instances were almost completely obliterated. The just described changes were less marked in the superior than in the inferior portion of the petrous tip. The dura of the posterior fossa had been removed at autopsy, and nothing can be said about its condition.

(*c*) Inner Ear: The lumen of the cochlea (fig. 10) was filled with fibrinopurulent exudate. The basilar membrane and Corti's organ were completely missing. The lower part of the basal coil was filled with granu-

g. 9 (case 5).—The vestibule is filled with granulation tissue. The annular ligament is almost completely yed, and the stapes is slightly dislocated. The area of the semicircular canals is occupied by a large abscess which contains sequestrated parts of the semicircular canals. *a.* indicates the abscess cavity; *e.d.*, the endo- atic duct; *n.b.*, newly formed bone; *n.VII*, the facial nerve; *r.o.*, rarefying osteitis; *s.c.*, a sequestrated part semicircular canal; *st.*, the stapes; *v.*, the vestibule, filled with granulation tissue.

ated the middle ear completely from the vestibule. anterior part of the stapes was slightly displaced rd the middle ear. The anterior-inferior circum- ce of the footplate showed marked osteoclastic n. There was some rarefying osteitis in both crura. cartilage covering the window frame was intact.

e deep layers of the cystic mucosa which filled iche of the round window have been transformed granulation tissue which extended through the win- into the basal coil of the cochlea. The round w membrane was completely destroyed.

) Mastoid Process: The remaining part consisted y of sclerotic bone. It contained a few small pneu- cells with thick, infiltrated mucosa and some w spaces filled with fat marrow, which showed is and extensive apposition of new bone to the lation tissue which, as described, went over into the granulation tissue of the round window. In some places a proliferation of the cellular elements of the endosteum was seen, with these growing into the exudate. The lowest part of the lamina spiralis ossea and the lamina spiralis secundaria were missing, and through the gap thus formed the granulation tissue extended into the vestibule. The enchondral capsule showed numerous resorption spaces with marked osteoclastic activity. In the upper part of the middle coil a large area of resorption was seen which extended through the entire thickness of the enchondral and endosteal capsules to the endosteum itself. The cells of the spiral ganglion and the nerve fibers were completely necrotic, and many spaces of the modiolus were filled with purulent exudate. The cochlear aqueduct in its entire course up to a point

not far from its inner aperture was filled with dense reticular tissue.

The vestibule (fig. 9) was completely filled with granulation tissue, which contained small pieces of necrotic bone. The membranous labyrinth was completely destroyed. In many places an osteoclastic erosion of the inner surface of the osseous wall was taking place. There was extensive rarefying osteitis in the surrounding labyrinthine capsule. In several places the capsule of the vestibule had been completely destroyed with formation of abnormal communications to the middle ear, the internal auditory meatus and the facial canal 5 mm. distal from the geniculate ganglion. Here the epineurium of the facial nerve was moderately infiltrated with round cells, but the nerve tissue itself showed no pathologic change. The gaps in the labyrinthine capsule were filled with granulation tissue, and the walls still showed osteoclastic destruction in some places while in others new formation of bone in a netlike arrangement was visible.

The vestibule went over posteriorly into a large cavity which occupied the site of the semicircular canals and communicated with the operative cavity (fig. 9). It was filled with pus and lined with a thick layer of granulation tissue. Its bony walls showed in many places apposition of newly formed bone in trabecular arrangement. The semicircular canals were extensively destroyed and partially sequestrated, with only p the lateral and superior vertical canals still recog The surrounding areas of the capsule showed a degree of rarefying osteitis. A larger cavity h formed by confluence of numerous resorption sp the retrolabyrinthine region, and its walls sho some places osteoclastic destruction. In other apposition of newly formed bone was noted. The lymphatic duct and sac were filled with fibrino exudate. The epithelial lining of the endolympha and sac was destroyed, and some parts of the wa necrotic. The surrounding bone showed ra osteitis, and some of the resorption spaces had into the vestibular aqueduct.

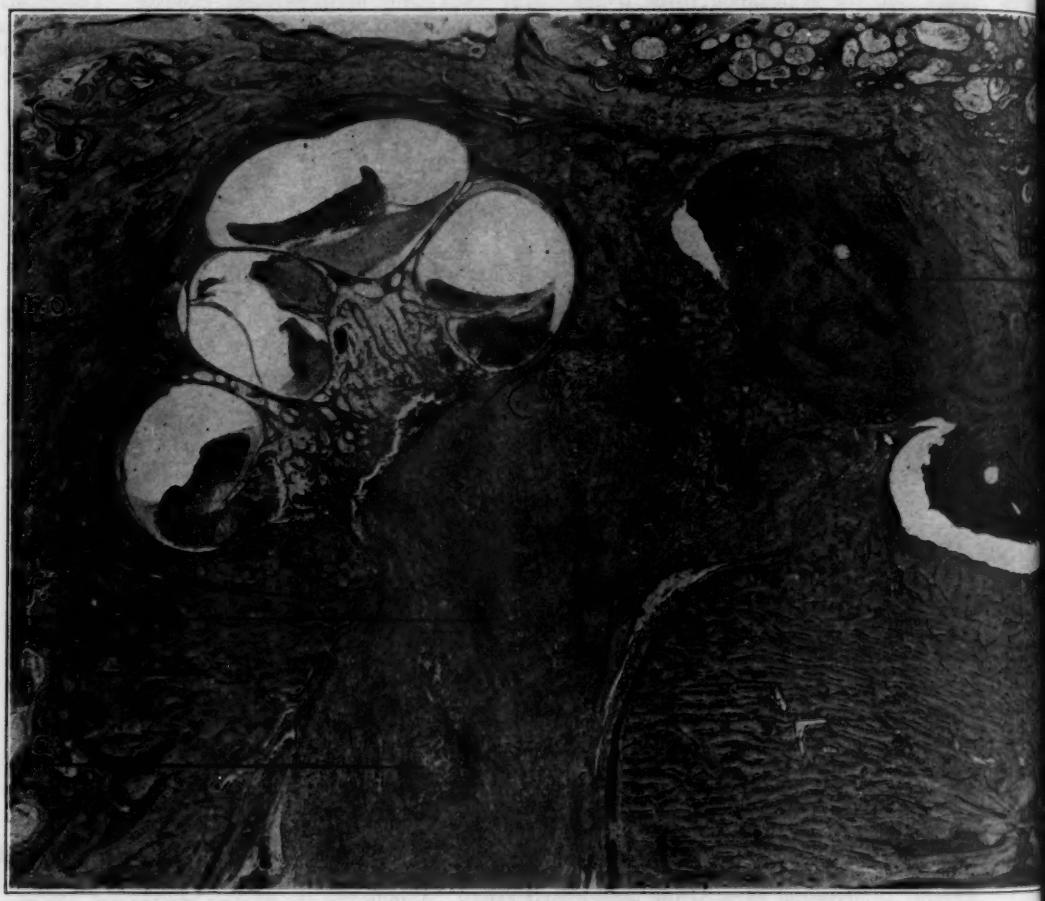


Fig. 10 (case 5).—Partially organized abscess of the fundus of the internal auditory meatus. The cochl filled with purulent exudate; the basal coil, with granulation tissue. There is marked rarefying osteitis cochlear capsule. *a.* indicates the part of the fundus abscess not organized; *b.c.*, the basal coil of the co *f*, the abscess which is being organized; *m.*, the cystic mucosa of the middle ear; *r.o.*, rarefying osteitis; vestibule.

The fundus of the internal auditory meatus con a large abscess which was being organized by g lation tissue (fig. 10). The eighth nerve was h infiltrated with granulation tissue. It was so thickened that it almost completely filled out the most part of the meatus. The nerve fibrils wer tensively destroyed. In some places, small abs were visible within the nerve itself and under arachnoidal sheath. The latter was infiltrated leukocytes in these areas. The facial nerve show slight amount of perineural infiltration with round but the nerve elements themselves seemed to be

logic Examination of the Right Temporal Bone. right temporal bone was essentially free from gic change.

ary.—Radical mastoidectomy had been performed year old diabetic woman for a running left ear years' duration. A fistulous opening was found region of the lateral semicircular canal; post- ely the patient showed signs of labyrinthine n for a few days. A month later the operative was revised because of recurrent attacks of dizzi- Three and a half months later, after a second of the operative cavity, the patient died of meningitis.

logic examination showed the labyrinthine cavi- ed with fibrinopurulent exudate and granulation There was extensive destruction of the laby- capsule in the region of the semicircular canals rtial sequestration of the latter. The other parts capsule showed extensive rarefying osteitis with on of abnormal communications between the ves- and the surrounding structures. The round membrane was completely destroyed, and granu- issue from the middle ear had grown into the mpani. There was partial destruction of the ligament, and the footplate of the stapes was dislocated, with granulation tissue from the yers of the covering mucosa growing into the e. The fundus of the internal auditory meatus d a large and partially organized abscess. From rganized parts of the latter, infection of the s had taken place. The primary infection of rinth had unquestionably taken place through the semicircular canal. At first labyrinthitis devel- a circumscribed area, causing recurrent attacks ness. At the time of the revision of the operative the labyrinth was apparently still functioning. are no data available about the time when the n became completely lost. In view of the exten- truction of the labyrinth it may safely be assumed time was shortly after the revision of the cavity. changes in both windows were so much less than those in the semicircular canals that un- nably they can be regarded as secondary, repre- reactive changes to the already existing infection labyrinth.

s case has been briefly discussed by E. P. Jr.[3])

COMMENT

ong the 5 cases just described the labyrin- infection had followed in 1 an acute puru- nfection of the middle ear, in 3 an acute rbation of a chronic infection of the mid- r of mesotympanic type and in 1 a chronic ion of the middle ear with cholesteatoma rosion of the lateral semicircular canal. In them the underlying infection of the middle the superimposed acute exacerbation had purulent (exudative) in character and d by hemolytic streptococci. In all the death was due to diffuse purulent labyrin- nic leptomeningitis.

4 cases the infection had spread from the e ear into the labyrinth through one or both windows; in 1 (case 5), through the semicircular canals.

The interpretation of the changes which accompany the spread through the windows is frequently not easy.

In many instances both windows are simultaneously affected, and sometimes the osseous capsule is also eroded in one or several places.

Under these circumstances, the lesions may represent multiple processes independently breaking through from the middle ear into the labyrinth, or one or several of them may be regarded as primary pathways of the infection and the others as secondary. They may represent either breaking out from the diseased labyrinth into the middle ear or secondary (dependent) breaking in (*zweite* or *abhaengigige Einbrueche,* to quote Lange [4]) from the middle ear into the labyrinth in response to stimuli emanating from the infected labyrinth into the surrounding tissues.

For the interpretation of a given case it is necessary to consider the histologic changes in the middle and the inner ear as a whole and to correlate them with the clinical course of the disease. Even then there will be great difficulties in many instances, and the final decision will depend to a great extent on the clinical history, the general impression of the case and the personal experience of the examiner.

For a better understanding of the changes involving the windows a thorough knowledge of the normal histology of the membranes of these windows seems indispensable.

The membranes of the windows are covered on the outer surface with a well vascularized layer of mucosa of the middle ear and on the inner surface with a thin and poorly vascularized endosteal layer. Between the two lies the main part, the membrana propria, composed of elastic and, to a lesser extent, of connective tissue fibrils. It is usually stated that the membrana propria is avascular (Zange [5]; Eckert-Mobius [6]) and that it is nourished mainly from the mucosal, to a much lesser extent from the endosteal, cov-

3. Fowler, E. P., Jr.: Suppurations of the Petrous A. M. A. **102**:1651 (May 19) 1934.

4. Lange, W.: Beitrag zur Pathologie der vom Mittelohr ausgehenden Labyrinthentzündungen, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **1**:31, 1908.

5. Zange, J.: Die pathologische Anatomie und Physiologie der mittelohrentspringenden Labyrinthentzündungen, Wiesbaden, J. F. Bergmann, 1919; Die konservative und chirurgische Behandlung der entzündlichen Erkrankungen des Innenohres mit besonderer Berücksichtigung der Indikationsstellung, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:1, 1927.

6. Eckert-Mobius, A.: Die pathologisch-anatomische Untersuchungstechnik und die normal histologischen Grundlagen, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 12, p. 1.

ering. This may be generally true, but the existence of anastomoses between the two vascular systems, extending through the membrana propria of the round window, has been demonstrated by Dean and Wolff.[7] The texture of the membrana propria of the round window is dense in the central parts and looser in the periphery. Here the fibrils diverge fanlike and radiate into the periosteum of the window frame. This is particularly marked in the lateral parts where the membrane is attached to the inner surface of the promontorial bone.

The annular ligament, according to the distribution of the elastic fibrils in the membrana propria, can be divided into three parts (Zange [5]). The external or periosteal layer, nearest to the middle ear, is rich, the middle layer poor, and the internal or endosteal layer moderately rich, in elastic fibrils.

The anatomic structure determines to a large extent the character of the changes which occur when a purulent infection progresses through the windows.

At first an infiltration of the membrana propria with leukocytes is noticed, and at that time bacteria and toxins might already have penetrated through the membranes and infected the labyrinth. In the round window membrane this infiltration occurs mainly in the peripheral parts, particularly near the attachment of the membrane to the inner surface of the promontory, where it is of looser texture. This is clearly seen in cases 2 and 4 of this paper. In the annular ligament the posterior part, near the facial canal, is often the most affected area. Sometimes, however, the posterior part is more resistant than the other parts—e. g., in case 5. The purulent infiltration is followed by decomposition of the elastic and connective tissue fibrils.

The destruction of the membranes always follows the same pattern, no matter whether the infection originates in the middle ear or in the inner ear. (Zange [5]). The inner layers are not as well nourished as the outer layers and are less resistant and more easily destroyed. This is particularly the case when the labyrinthitis has already developed and the flow of nutrition from the inside has been completely suspended. (See annular ligament in cases 1 and 5.) In many cases of acute purulent infection and in most cases of subacute and chronic infection the mucosal covering remains intact for a long time or even permanently. The mucosal covering is eventually perforated. The destruction of membrana propria is followed by the for of a gradually growing intramucosal a which finally opens into the tympanic ca into the labyrinth—e. g., in case 1, into th tympani.

The marked tendency toward preserva the mucosal covering is clearly seen i changes which follow complete destruction annular ligament. In case 1, only the inne of the ligament was destroyed, while the parts and the mucosal covering were d infiltrated. Case 2 must be omitted beca secondary traumatism. In case 5 the an ligament was destroyed to a great exten in case 3 it was completely destroyed. N theless, the continuity of the mucosa had nowhere interrupted. Its deep layers had transformed into granulation tissue. E case 4, in which the stapes had completel appeared, the middle ear was completely rated from the vestibule. Apparently aft disappearance of the stapes the granulatin edges of the mucosa had grown togeth analogy to the formation of a neomembra the external auditory meatus. This "neo brane" had then been covered with n epithelium by extension of the latter fro periphery toward the center. The granu tissue which originates in the deep layers mucosa may grow into the vestibule. Som during this ingrowth an erosion of the w frame takes place (case 4). In 3 cases the footplate of the stapes was slightly rotated a a vertical axis and its anterior part som dislocated toward the middle ear (cases 1, 5). A similar position of the footplate wa quently seen by Zange.[5] It occurs in ca which the mucosal covering and the sta muscle and tendon are intact and is appa due to the pull of the muscle on the stapes, becomes manifest when the counteraction annular ligament is eliminated. It is marked when the annular ligament is more destroyed (cases 2 and 5) but is appa possible also when the resistance of the lig is only weakened (case 1).

In the round window the initial peri cellular infiltration (case 2) is followed by the formation of an abscess (case 1) o further extension of the infiltration and appearance of capillaries within the infil areas (case 4). We must assume that m these capillaries are newly formed and d from vascular buds originating in the layers of the covering mucosa and growing and through the membrane itself. Some

7. Dean, L. W., and Wolff, D.: Pathology and Routes of Infection in Labyrinthitis Secondary to Middle Ear Otitis, Ann. Otol., Rhin. & Laryng. **43**:702, 1934.

s, however, might be preformed (Dean and
"). This progressive infiltration of the
rane may lead to its gradual extensive
3) or even complete destruction (case 5).
the cellular and vascular infiltration has
d the inner surface of the membrane, the
helial covering is, as a rule, extensively
ed by the labyrinthine infection. The
tep is an ingrowth of connective tissue,
advances into the scala tympani and from
into the innermost part of the aqueductus
ae (case 3); after the destruction of the
spiralis secundaria and of the adjoining
of the basilar membrane, the connective
extends into the vestibule (cases 4 and 5).
inability to recognize from the histologic
e alone the direction from which the infec-
as penetrated the membranes and the dif-
in differentiating between primary and
dary pathways of infection in the already
bed sense make it well understandable that
questions regarding the genesis of labyrin-
in a given case may remain controversial.
has been claimed that hyperplastic-cystic
al changes in the window niches facilitate
n determine the localization of a labyrin-
infection by interfering with the free drain-
pus toward the middle ear. As a matter
t, hyperplastic mucosal changes were pres-
n all our cases. According to Zange,[5]
ver, retention of pus plays only a minor role
npared with that of large numbers of highly
nt germs, particularly when the infection
nbined with a lowering of the general resis-
of the body.

ny authors have expressed the belief that
ound window represents a more frequent
of entry than the oval window and that
both windows are affected the round one,
ule, is more severely diseased than the oval
w. Zange,[5] after a careful analysis of 228
denied the greater frequency or severity
ections of the round window.

ong the 4 cases of window labyrinthitis
bed in this paper the infection occurred
rily through the round window once (case
rimarily through the oval window twice
s 2 and 4) and probably through the oval
w but possibly through both windows
taneously once (case 3).

yrinthine infections arising solely through
istent vascular connections or normal ana-
fissures of the capsule, such as the fissula
enestram or the fissure between the niche
e round window and the ampulla of the
ior vertical canal, are undoubtedly extreme-
re. A concomitant infiltration of these areas, however, in cases in which the main infection has gone through other portals of entry, is much more common than one would think from the scarcity of reports in the literature. Among our cases, both fissures were infiltrated in case 2.

Although the underlying infection of the middle ear in all our cases had been purulent, the labyrinthitis in 3 cases presented the characteristic picture of a so-called primary purulent-necrotizing inflammation (Zange[4]) throughout the labyrinth; in case 3 there was a simple purulent infection in the upper part of the cochlea while in the other parts the infection was of a purulent-necrotizing type. These observations show that the character of the labyrinthine infection is determined by factors other than the character of the infection of the middle ear, such as lesser resistance of the structures of the inner ear or flooding of the inner ear with highly virulent germs together with lowering of the general resistance of the body. This can also be seen from the fact that necrotizing infections of the middle ear in scarlet fever and in tuberculosis rarely cause necrotizing labyrinthitis.

Reactive reparatory changes start shortly after the onset of the labyrinthine infection. As already stated, the histologic changes observed in the window membranes in the reparatory stage are practically identical with those seen during the primary invasion of the labyrinth, and this is one of the difficulties in the proper interpretation of certain cases. No matter whether the chemotactic stimuli which eventually lead to the destruction of the membrane emanate from the middle ear or from the labyrinth, the leukocytes which destroy the membranes emigrate mostly from the well vascularized mucosal covering on the lateral surface and not from the poorly vascularized medial surface of the membrane. In purulent infections of the labyrinth the changes just mentioned are accompanied or followed by proliferation of the endosteum with formation of organization tissue, which grows into the exudate (cases 3 and 5). In the scala tympani of the cochlea the connective tissue within Rosenthal's canal participates in the formation of the organization tissue (case 3).

In purulent-necrotizing inflammations granulation tissue may grow into the labyrinthine cavities from the neighborhood, particularly through the windows or from the fundus of the internal auditory meatus into the basal cochlear turn, especially in those cases in which an abscess of the fundus is being organized (cases 4 and 5). Owing to the extensive destruction or necrosis of the endosteum, the organization of the exudate is aided by granulation tissue which

originates in the capsule. At first an area of rarefying osteitis is seen in the capsule, and by gradual extension and confluence of the perivascular resorption spaces larger foci are formed, which eventually reach the labyrinthine cavities and from which an ingrowth of granulation tissue takes place. By outward extension of the same foci, abnormal communications between the labyrinthine cavities and the neighborhood may be established. The resorption spaces, which in the beginning are free from infection, become secondarily infected as soon as they come in contact with the exudate within the labyrinth. In the course of the osteitic changes in the capsule, larger parts of it may become completely surrounded by granulation tissue and sequestrums formed (case 5). In cases of purulent infection the process of healing when not interrupted by the development of a deadly intracranial complication or by operative intervention terminates in complete organization of the exudate with extensive or complete obliteration of the lumen and at least partial ossification of the organizing tissue. In cases of purulent-necrotizing infection this is usually accompanied by partial destruction of the capsule.

The successful use of chemotherapy in the prevention and the treatment of labyrinthogenic endocranial complications will undoubtedly result in an increase in the number of cases of healed labyrinthitis, and a thorough knowledge of the histologic changes of this condition seems important.

Although the question of the pathways of the endocranial extension of the infection has lost some of its practical importance, it still deserves close attention. No matter how great the therapeutic progress will be, one can hardly conceive that therapy will succeed in completely eradicating those complications. The unfavorable conditions of drainage which result from the peculiar configuration of the labyrinth and which unquestionably facilitate the development of meningitis are still aggravated by the already discussed tendency of the mucosal covering of the window membranes to stay intact for a long time, even permanently.

The main pathway of infection leads through the channels of the modiolus into the fundus of the internal auditory meatus (cases 1, 2, 4 and 5), and frequently the nerve and vascular channels which lead into the vestibule and to the ampullas are also affected (cases 1, 2 and 4). From the fundus the infection may spread directly to the leptomeninges (cases 1 and 2) or may be temporarily (cases 4 and 5) or even definitely halted by the formation of an abscess in the arachnoidal cisterna of the fundus (mann and Waltner [1]). The delaying acti clearly seen in the described cases in which occurred two and a half to three and a months after the first signs of purulent lab thitis, compared with fifteen days in ca These cases illustrate clearly the dangers o so-called "semilatent" or early latent sta chronic purulent labyrinthitis and the diffic arising in the management of such cases. abscess of the fundus the blood supply to inner ear is shut off in certain cases and a plete, so-called secondary, necrosis of the parts of the labyrinth takes place (secon necrotizing labyrinthitis—Zange [5]). Emp of the endolymphatic sac is by no means un mon (cases 1, 2, 3 and 5), but invasion of meninges from there by way of an interca intradural abscess is rare. It was seen in ca but there was a simultaneous invasion of leptomeninges from two deep epidural absc of the posterior fossa which had developed diseased retrolabyrinthine cells. The coc aqueduct plays only a minor role as a path The cases described in this paper confirm general rule that the genesis of labyrinthiti determined by the character of the underl infection of the middle ear. If the latter is dominantly exudative (cases 1 and 4), the in tion will, as a rule, spread through the windo If it is predominantly proliferative pneumoco type III infection, it will progress through osseous capsule, particularly through the s circular canals. This is true also in case chronic otitis media with cholesteatoma (5). In cases of acute exacerbation of chr suppuration (cases 2 and 4) it will spr through the windows, but in those cases in wh a labyrinthine fistula has been present before development of the exacerbation, it will penet through the fistula.

Although hemolytic streptococcus infec almost invariably causes exudative inflammat the term "exudative otitis" does not necessa denote an infection with that germ, for other teria show a similar mode of action.

SUMMARY AND CONCLUSIONS

Hemolytic streptococci were the cause of t panogenic labyrinthitis in the 5 cases descri Labyrinthogenic meningitis was the cause death in all of them. In 1 case the labyrinth had followed acute otitis media, in 3 an ac exacerbation of chronic otitis media and in chronic otitis media with cholesteatoma.

In acute exudative otitis media or in ac exacerbations of uncomplicated chronic o

spread into the labyrinth occurs, as a rule, h one or both windows. It is questionable er the round window or the oval window re frequently the portal of entry for the on. In the cases described in this paper, fection penetrated once through the round w, twice through the oval and once prob- through the oval window but possibly h both windows simultaneously.

an analysis of the changes in the window ranes in the light of the peculiarities of the mic structure of these areas, it is pointed out he histologic changes occurring in a primary ion of the windows are identical with the dary reactive changes following a purulent ion of the labyrinth through another portal try. This is one of the main difficulties in roper interpretation of some of the cases yrinthitis.

e mucosal covering of each of the window ranes shows a marked tendency to remain even after complete destruction of the rest e membrane. This undoubtedly impedes rainage toward the middle ear and facilitates pread toward the leptomeninges.

e frequently encountered rotation of the s around a vertical axis after extensive or lete destruction of the annular ligament is ined by the physiologic action of the stapedial muscle, which in these cases had remained intact.

The genesis of labyrinthitis is largely determined by the character of the underlying infection of the middle ear. Exudative inflammations penetrate through the windows; proliferative infections (pneumococcus type III) and chronic infections complicated with cholesteatoma, through the osseous capsule, particularly the semicircular canals.

The character of the labyrinthitis, however, is not determined by the character of the otitis media but by other factors. Although in the cases of this paper the infection of the middle ear was purulent, the following labyrinthitis was purulent-necrotizing in character.

Among the pathways of an extension of the infection to the meninges, the spread along the channels of the modiolus and the vestibule is by far the most important. Empyema of the endolymphatic sac and perisaccular abscess play only a minor role. A deep abscess of the fundus of the internal auditory meatus may delay or even prevent the spread to the meninges. Among the cases described in this paper, the meningitis had four times developed through the modiolus and had twice been halted temporarily by a deep abscess of the fundus. Once it had developed from a perisaccular empyema.

PHYSIOLOGIC EFFECT OF PRESSURE CHANGES WITH REFERENCE TO OTOLARYNGOLOGY

COMMANDER A. R. BEHNKE (MC), U.S.N.

The remarkable phenomenon of adaptation to environmental stress is exemplified by man's tolerance of rapid and extreme alterations in barometric pressure. If pressure per se is considered apart from the effect of gases in the viscera, in aural and sinal spaces and in solution in tissues, then variations in pressure in the range of 0.11 atmosphere (50,000 feet [15,000 meters] altitude) to 16.1 atmospheres (500 feet [150 meters] diving depth) are without physiologic effect.

COMPRESSION

When the body is subjected to increased pressure, every air space, from the smallest and most inaccessible ethmoid cell to the air cells in the mastoid process, receives air provided that the passages to these spaces are unobstructed. The ingress of air to the aural spaces must be consciously brought about by various maneuvers, such as swallowing, yawning and using the Valsalva maneuver, which relax tension on the normally closed "flutter valve" of the eustachian tube.

The rate of accommodation to increasing pressure in experienced persons in diving operations may be as rapid as 45 pounds per square inch (2,280 mm. of mercury) per minute, which is equivalent to a diving descent at the rate of 100 feet (30 meters) per minute. In routine training operations, in which factors other than the ability to equalize intratympanic pressure are present, the average time of descent to a depth of 225 feet (68 meters) by student divers making four hundred dives was five and two-tenths minutes, with a range of two to fourteen minutes (table 1). In similar dives with helium-oxygen mixtures being used, the average time of d to 225 feet in four hundred dives was fou six-tenths minutes, with a range of two to f minutes. For deep sea dives by experie men (U.S.S. *Squalus* operations), the av time was about three and five-tenths minute depths of 225 to 240 feet (68 to 72 me These average values ranging from one and tenths to three and seven-tenths minutes pe feet do not represent maximal rates but r rates that are well tolerated by healthy me rates that do not produce trauma of sinal aural membranes.

TABLE 1.—*Rate of Accommodation of the Aural Sinal Spaces to Increased Pressure*

Diving Operations							
			Pressure (Gage)		Time (Min.)		
Breathing Medium	No. of Dives	Depth, Ft.	Lb./ Sq. In.	Mm. Hg	Total	Range	Comm
Air	301	125	56	2,879	4.7	1-17	Student d
Air	400	225	100	5,182	5.2	2-14	(wet tank
He+O2	400	225	100	5,182	4.6	2-15	Open sea
He+O2	145	225	100	5,182	3.7	2-7	(U.S.S.
		to 240	107	5,527			

Altitude Descent			
Breathing Medium	Simulated Altitude, Ft.	Pressure, Mm. Hg	Re
O2	48,000 to 12,000	96 to 483	
O2	45,000 to 15,000	111 to 429	
O2	45,000 to 12,000	111 to 483	
O2	41,000 to 10,000	134 to 523	
O2	40,000 to 10,000	141 to 523	
O2	40,000 to sea level	141 to 760	
O2	40,000 to sea level	141 to 760	

With respect to high altitude, simulated scents from 40,000 feet (12,000 meters) to level have been made in the low pressure cha in thirty-seven seconds without injury (table In routine experimental simulated descents, average rate of descent was 5,000 feet (1 meters) per minute. This figure stands in c trast to the rate of 300 feet (90 meters) per ute stipulated for commercial aviation to mini the occurrence of aero-otitis media.[1]

It would appear from the data presented less time is required for compression from

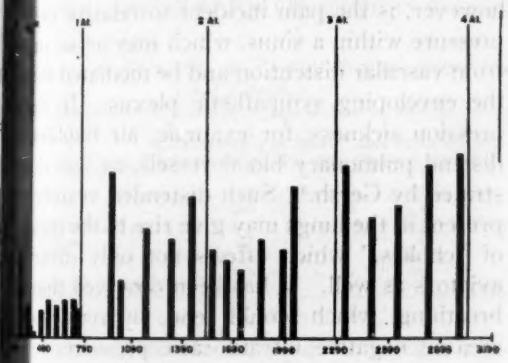
From the United States Naval Medical Research Institute.

This paper was presented at the annual meeting of the American Academy of Ophthalmology and Otolaryngology in Chicago, Oct. 8, 1944.

This article has been released for publication by the Division of Publications of the Bureau of Medicine and Surgery of the United States Navy. The opinions and views set forth are those of the writer and are not be be construed as reflecting the policies of the Navy Department.

1. Armstrong, H. G.: Principles and Practice Aviation Medicine, Baltimore, Williams & Wil Company, 1939.

mosphere than from 1 to 4 atmospheres. umber of deglutitions required by divers alize pressure in descent from 34,000 feet, 360 meters, (0.25 atmosphere) to ground nd from the surface to a diving depth of eet (4 atmospheres) stands in the ratio o 13. If pressure is uniformly increased 0.25 to 1 atmosphere during a period of six es and then after a single fifteen minute l at ground level increased to 4 atmos-, it is observed that the differences in pres- ncrease despite the fact that complete equili- n of pressure in the tympanum is apparently fected by single acts of swallowing (fig. 1). n greater differences in pressure are toler- f stops are prolonged at each equilibration o insure complete equalization of pressure ans of the Valsalva maneuver in addition to wing. A difference in pressure, for example, mm. of mercury might require deglutition



1.—Differential pressures requiring the act of wing for tympanic equilibration in continuous ted descent from 33,500 feet, or 10,307 meters, atmosphere) to ground level (1 atmosphere) in inutes and from 1 to 4 atmospheres (99 feet [30 s] depths) in six minutes.

5 atmosphere (18,000 feet [5,400 meters]), t 4 atmospheres (100 feet diving depth) the sponding difference might be 350 mm. of ury (fig. 2).

hus, over a wide range of pressure, the toler- of the tympanum to differences in pressure ore closely related to a constant change in me than to a constant difference in pressure. ncrease in pressure, for example, from 1 to mospheres tends to have an effect equivalent increase from 2 to 4 atmospheres, since the ge in volume of intra-aural air is the same r both conditions. The problem of whether t constant difference in pressure or a volume governs the rate of compression is some- complicated by the increasing density of ccompanying the augmented pressure. To evaluate the factor of density, experiments were performed in which a helium-oxygen mixture was breathed in place of air as the pressure was raised from 1 to 4 atmospheres. The results showed that the rate of voluntary opening of the auditory tubes was not influenced by the density of the

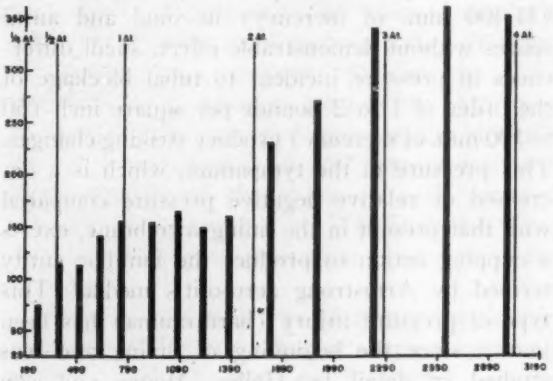


Fig. 2.—Differential pressures requiring tympanic equilibration during descent from 33,500 feet (10,307 meters) to ground level and during compression from 1 to 4 atmospheres. At each level stops were made to insure complete equilibrium of pressure through performance of the Valsalva maneuver and swallowing.

gas breathed (table 2). Subjects reported, however, that equalization of pressure was easier when they breathed the helium-oxygen mixture.

TABLE 2.—*Pressure Differences (in Millimeters of Mercury) at Which Equalization of Pressure Was Effected by Swallowing When the Barometric Pressure Was Uniformly Raised From 760 to 3,020 Millimeters of Mercury (One to Four Atmospheres) in Eight Minutes*

	Air Inhalation, Atmospheres			Helium-Oxygen Inhalation, Atmospheres		
	1 to 2	2 to 3	3 to 4	1 to 2	2 to 3	3 to 4
	91	91	104	61	110	112
	81	89	102	62	94	111
	51	76	98	71	80	109
	76	99	127	107	99	132
	81	97	105	71	88	102
	81	100	137	99	107	75
	82	86	107	76	107	104
	93	130	...	79	99	...
	86	..	...	89	..	...
Average	80.2	96	111.4	79.3	99.1	106.4

DECOMPRESSION

In contrast with the effects of compression, the release of air pressure from patent sinal and aural cavities can take place almost instantaneously without giving rise to abnormal symptoms.

Explosive decompressions simulating altitude increments of 20,000 feet and which tend to create excess pressure of 3.5 pounds per square inch (181 mm. of mercury) may be effected in a fraction of a second. Diving ascents likewise can

be made from depths of 100 feet in less than thirty seconds.

SYMPTOMS PRODUCED BY DIFFERENCES IN PRESSURE

Although air pressure has been increased to values as high as 225 pounds per square inch (11,400 mm. of mercury) in sinal and aural spaces without demonstrable effect, small differences in pressure incident to tubal blockage of the order of 1 to 2 pounds per square inch (50 to 100 mm. of mercury) produce striking changes. This pressure in the tympanum, which is a decreased or relative negative pressure compared with that present in the lining membrane, exerts a cupping action to produce the familiar entity termed by Armstrong aero-otitis media. This type of pressure injury (barotrauma) has been known since the beginning of diving and was studied in detail by Heller, Mager and von Schrötter,[2] who noted the hyperemia ex vacuo, congestion of the membranous lining of the middle ear and tympanic membrane, hemorrhage and, occasionally, perforation of the membrane. The pathologic picture, depending on the difference in pressure, shows, progressively, retraction of the tympanic membrane, appearance of blood vessels in Shrapnell's membrane and malleolar area and their subsequent dilation, engorgement and rupture to produce exudation of fluid, hemorrhage into the middle ear and, finally, rupture of the tympanic membrane. The condition, except for retraction of the tympanic membrane, may be indistinguishable from an acute infectious otitis media.

The cupping action affects not only the tympanic membrane but also the whole membranous lining of the middle ear and the mastoid cells. Similar changes take place in membranes lining obstructed sinuses.

The important physiologic principle is that a difference in pressure between the intra-aural, intrasinal space and the enveloping membrane which is under the pressure of ambient air elicits pain. With reference to the ear, pain is felt deeply within the ear and over the parotid and mastoid areas, which may be tender to palpation. If the tympanic membrane of the occluded ear is inspected as the ambient pressure is increased, it will be noted that retraction of the membrane and appearance of blood vessels precede pain. The sinal pain is superficial and is distinguished by a sensation of multiple lancinating stimu by its sharp limitation to the affected area. a diagnosis of closure of the maxillary, et or frontal sinus is evident. The sphenoid s alone appear to be free from obstructive im ment. Palpation of the affected areas elici derness.

Occasionally, during decompression, th stachian tube may be blocked, and an excruc type of pain may be produced in the midd as the tympanic membrane is pushed outwa the relative positive pressure in the tymp A similar obstruction of a sinal ostium, u the frontal, likewise elicits pain, which, how is diffuse, dull and aching in contrast wi lancinating pain associated with negative pressure.

The cause of pain associated with relative tive pressure in sinal spaces is obviously r to compression of the lining membrane a the osseous wall. On a wholly different however, is the pain incident to relative neg pressure within a sinus, which may arise, in from vascular distention and be mediated thr the enveloping sympathetic plexus. In de pression sickness, for example, air bubbles distend pulmonary blood vessels, as was dem strated by Gersh.[3] Such distended vessels present in the lungs may give rise to the cond of "chokes," which affects not only diver aviators as well. It has been observed that breathing, which would tend, by reason d creased negative intrathoracic pressure, to ment vascular distention, elicits the presen early or otherwise unrecognized "chokes."[4]

These observations, in summary, indicate differences in pressure of small magnitude, w occasionally compress sinal and aural membr and usually give rise to a cupping action, b about vascular distention, congestion and he rhage associated with pain restricted to and d nating the area of involvement.

Oxyotitis Media.—In 1940, divers ma simulated altitude ascents to 38,000 feet (11 meters) returned to ground level entirely from objective or subjective aural sympt During the night or on awakening the follo morning, they complained of earache, a sens of fulness and especially of fluid in the ears impaired hearing. Inspection of the tymp membranes usually revealed changes simila

2. Heller, R.; Mager, W., and von Schrötter, H.: Luftdruck-Erkrankungen, mit besonderer Berücksichtigung der sogenannten Caissonkrankheit, Vienna, A. Hölder, 1900, vols. 1 and 2.

3. Gersh, I.: Personal communication to the au

4. Behnke, A. R.: High Atmospheric Pressu Physiological Effects of Increased and Decreased P sure; Application of These Findings to Clinical M cine, Ann. Int. Med. **13**:2217-2228, 1940.

found in the early stages of aero-otitis [5]

the spring of 1942, similar complaints were by engineers engaged in repeated high alti- flights. The symptoms were at times suffi- y severe to produce disability.

e explanation of the puzzling interval of being followed by delayed symptoms inci- to sleep is found in the effect of negative ure brought about by the absorption of oxy- from the middle ear spaces during sleep, conscious opening of the eustachian tubes is essed. In ascent to 38,000 feet (11,692 rs), for example, four fifths of the air origi- present leaves the middle ear. During nt to ground level, if oxygen is inhaled dur- he whole period, the middle ear becomes with oxygen in high concentration. The ure of oxygen in the tympanum is of the of 600 mm. of mercury, while the pressure ygen in the tissue may be less than 40 mm. ercury. Owing to the utilization of oxygen,

3.—*Gas Pressures in Tissues and in Aural and Sinal Cavities*

	Gas Pressures in Sinal, Aural Cavities, Mm. Hg		Gas Pressures in Tissues, Mm. Hg
gen	568	←	573
n	150	→	40
on dioxide	...	←	47
r vapor	47		47
	760	→	707

pressure in the tissue tends to remain con- . The resultant high pressure gradient main- diffusion of oxygen out of the space, and a cupping effect is exerted on the mem- ous lining. The diffusion of nitrogen out of tissues apparently is too slow to compensate he loss of oxygen. A possible local irrita- action of oxygen has not been evaluated.

he familiar "vacuum" frontal headache and changes induced by the absorption of oxygen complete tubal obstruction have the same ogic basis.

he fact that relative negative pressure in the ille ear spaces can give rise to pain in the toid region and tenderness when the area is ated emphasizes the need to consider a type nastoiditis caused by obstruction rather than infection. The consideration is applicable cally to cases of catarrhal otitis media, cially in children, in whom the more abun- dant epipharyngeal lymphoid tissue can more frequently obstruct the auditory tube to produce "aeromastoiditis."

The values shown in table 3 are representative of gas pressures in tissues and in ventilated sinal and aural cavities.

After occlusion of a sinal ostium, the pressure tends to decrease to 707 mm. of mercury. Since the pressure in the blood vessels is 760 mm. of mercury plus capillary pressure, the blood vessels begin to dilate as soon as the absorption of oxygen in the occluded space results in a falling off of pressure from 760 mm. of mercury. Measurements of the order of — 25 mm. of mercury were obtained by Van Dishoeck [6] and were attributable to resorption of oxygen in the tympanum.

Prevention of this type of injury consists in breathing air during descent, beginning at an altitude of 15,000 feet (4,615 meters). In the comprehensive studies of Bowen,[7] air inhaled at still higher levels during descent was found to be even more effective.

Effect of Variations in Pressure and of Trauma on Hearing.—Confusion exists with reference to impairment of hearing induced by pressure trauma. Following acute trauma, the audiogram reflects diminished perception of sound over the whole frequency range. As the pathologic disturbance undergoes resolution, however, hearing returns to the initial level of acuity. The rarity or absence of proved cases of deafness arising from injury incident to pressure trauma in deep sea diving or caisson work stands in contrast to the permanent damage to the ear caused by gunfire.

Heller, Mager and von Schrötter stressed the fact that permanent deafness from compression injury did not occur. These authors clearly distinguished between compression injury of the tympanum and Ménière's syndrome, which was not of infrequent occurrence (14 cases among 675 workers) and was thought to be due to nitrogen bubbles interfering with the blood supply of the cochlea. This concept is supported by the fact that there is a delay of the appearance of the syndrome after decompression, that it rapidly disappears spontaneously or during recompression, that there is frequent absence of changes in the middle ear and that the syndrome never occurs during compression.

, Behnke, A. R.: Investigations Concerned with lems of High Altitude Flying and Deep Diving: lication of Certain Findings Pertaining to Physical ess to the General Military Service, Mil. Surgeon -28, 1942.

6. van Dishoeck, H. A. E.: Measurement of the Tension of the Tympanic Membrane and of the Resistance of the Eustachian Tube, Arch. Otolaryng. **34**: 596-602 (Sept.) 1941.

7. Bowen, W. J.: Delayed Acute Aero-Otitis Media and Methods of Prevention, U. S. Nav. M. Bull. **44**: 247-252 (Feb.) 1945.

Two cases, however, have been reported by Simpson [8] in which Royal Air Force aviators became permanently deaf after rapid descents incident to dive bombing. McGibbon,[9] in a study of 100 aviator patients with symptoms referable to the ear due to too rapid compression, reported that although deafness was the most common complaint not one of the series manifested a permanent "inner ear" type of deafness mentioned by Simpson.

Of critical importance is the matter of possible injury to the labyrinth. Heller, Mager and von Schrötter cited the measurements by Politzer and Bezold, which indicated that a decrease in labyrinthine pressure accompanied the decrease of relative pressure in the tympanum. They believed that stasis, transudation and hemorrhage occurred in the labyrinth as well as in the cleft of the middle ear.[2] In the absence of permanent deafness, however, it does not appear likely that such destructive changes affect the labyrinth.

It is to be expected that the pressure in the labyrinth will equal or closely approximate that of the cerebrospinal fluid, which attains equilibrium with variations in ambient pressure without a lag. Under these conditions, the integrity of labyrinthine structure would seem to depend on the resistance of the round window to any relative negative pressure existing in the tympanum.

In 3 dogs subjected to rapid compression, Vail [10] found that although the tympanum was severely injured there were no appreciable alterations in the labyrinth. Additional studies are required to settle this critical problem.

With respect to deep sea divers subjected to varying degrees of pressure trauma, usually of mild degree over a period of five to fifteen years, audiograms indicate that the loss of hearing at 4,096 double vibrations is consistent with the average loss for a similar control group.

The impressive fact, however, is the paucity of cases of permanent deafness attributable to barotrauma, not only in deep sea divers but in aviators as well.

Incidence of Aero-Otitis Media and Aerosinusitis.—The incidence of aero-otitis is primarily related to the degree of tubal obstruction and the rate at which the compressive force is applied. The structure of the eustachian tube makes it peculiarly susceptible to obstruc The vascular mucosa, filled with mucin-secr goblet cells and supported by a rich vas and glandular bed containing lymphatic t lines a firm cartilaginous tube, which easil comes sealed by edema, cellular infiltration mucus. The presence of lymphoid tissue a and within the opening acts as a potential It is not surprising, therefore, that infec allergic response and such mild trauma as induced by negative pressure, singly or in co nation, produce frequent obstruction.[11]

The factor of trauma arising from neg pressure is better appreciated when one siders that after successive exposures to pre the equalization of pressure may become difficult, owing to congestion and edema fro continual effort expended to "clear the ear

The degree of tubal obstruction depends ly, however, on the presence and severity o fection in the nasopharynx. Colds represen most common type of infection causing disab It is not surprising, therefore, that the inci varies from 5 to 20 per cent in diving and operations. In 1932, of 453 submarine perso 11.5 per cent were unable, because of tubal b age, to equalize 50 pounds of pressure per sq inch (2,533 mm. of mercury) applied in a cha during a period of five to ten minutes. F tenths per cent of the men suffered from blockage. In 1936, out of 1,748 submar personnel, 8 per cent subjected to the same sure test were disqualified because of tubal b age.[12] In a pressure chamber test for sta divers, 60 men out of 326 failed on their attempt because of colds, but only 8 of the p were permanently disqualified.

VALUE OF A PRESSURE CHAMBER AS MEANS OF DIAGNOSIS

During the past fourteen years pressure 5 to 50 pounds per square inch (253 to 2,533 of mercury) applied to candidates for subma escape drill and deep sea diving have dem strated the value of the pressure chamber means of diagnosis of aural and sinal block Its obvious advantages consist chiefly in the a cation of a wide range of pressures at a contr rate to 1 or more persons.

8. Simpson, J. F.: A General Survey of Otorhinological Considerations in Service Aviation, J. Laryng. & Otol. **57**:1-10, 1942.

9. McGibbon, J. E. G.: Aviation Pressure Deafness, J. Laryng. & Otol. **57**:14-22, 1942.

10. Vail, H. H.: Traumatic Conditions of Ear in Workers in Atmosphere of Compressed Air, Arch. Otolaryng. **10**:113-126 (Aug.) 1929.

11. Simkins, C. S.: Functional Anatomy of Eustachian Tube, Arch. Otolaryng. **38**:476-484 (1943. Graves, G. O., and Edwards, L. F.: The tachian Tube: Review of Its Descriptive Micros Topographic and Clinical Anatomy, ibid. **39**:35 (May) 1944.

12. Behnke, A. R.: Submarine Medicine, Mil geon **83**:6-18, 1938.

the Navy, it has served to indicate the ence of nasopharyngeal infection which rily would not exclude a man from duty. excepting those with patulous membranes, an accommodate pressure at the rate of 45 s per square inch (2,280 mm. of mercury) nute are comparatively free of nasopharyn- nfection. Some 30 to 50 per cent of a of apparently healthy persons may be ed to fail in this test at any given time. If e of compression is extended over a period minutes, then from 5 to 20 per cent of a group of men will fail in the test.

degree of tubal obstruction is reflected tatively in the rate of tolerance to increased re. A person with a cold may require a of thirty minutes to accommodate to an pressure of 5 pounds per square inch mm. of mercury). The empiric data point igh correlation between acute nasopharyn- fection and tubal obstruction. The relation n subclinical infection of the pharynx and blockage merits further study.

THERAPY

servative treatment in the form of non- tic ventilation and drainage can be carried the application of controlled pressure at a lerated by the patient. Both inspection of mpanic membrane and symptoms of dis- t serve to protect against injury from too application of pressure. The release of t pressure after it has been raised to 5 or nds per square inch (253 to 507 mm. of ry) will serve to effect drainage without , since intra-aural or intrasinal pressure become negative.

following statements outline the responses ients subjected to pressure and illustrate les underlying diagnosis and therapy [13]:

1.—The complaint was that of headache over s and eyestrain. In the middle meatus of each polyps were present. The pressure, after being o 10 pounds per square inch (507 mm. of mer- n the compression chamber, was rapidly re- Inspection of the left meatus during the release ure revealed a bubbling out of air and secretion neath the polyps.

2.—Pain, intensified by the patient's bending as present over the eyes. Errors of refraction t found. The nasal septum was deviated to the touched the middle turbinate. Purulent secre- s present. The tympanic membranes appeared After the slow application of pressure, pain ared. On release of pressure, however, severe veloped over the eyes. Again the pressure was raised to 15 pounds (760 mm. of mercury) and the pain was relieved, only to return when the pressure was released. An astringent was then applied to the nasal mucous membranes. Purulent secretion and air bubbles were observed to exude from the opening of the fronto-nasal duct accompanied with a swishing sound. With the equalization of pressure, pain was relieved.

Comment.—This case illustrates the relation of symptoms to inequality of pressure. Intrasinal pressure either higher or lower than pressure in the tissue was associated with pain.

CASE 3.—Frontal pain on bending over, malaise and nasal congestion were present. Pressure was raised to 2 pounds (100 mm. of mercury) and was accompanied with intense, lancinating, multiple stimuli pain in the left eyebrow and nostril, which slowly subsided. Pressure was raised to 5 pounds (253 mm. of mercury) and then decreased slowly. The release of pressure was accompanied with serous drainage. On successive days the pressure was gradually increased. With the subsidence of infection, the usual rate of increase in pressure was accommodated.

CASE 4.—The patient had a head cold, with malaise, nasal congestion and frontal pain on lowering the head. Pressure was raised to 2 pounds (100 mm. of mercury) in the chamber. Intense lancinating pain was felt over the left eyebrow and in the left nostril. The pain slowly subsided as the pressure was equalized. The pressure was raised to 5 pounds (253 mm. of mercury) and then decreased slowly to produce a small amount of serous drainage.

The next day the pressure was raised to 7 pounds (355 mm. of mercury). Intense pain was experienced over the left eye and bridge of the nose at 3, 5 and 7 pounds of pressure (152, 253 and 355 mm. of mercury). On rapid release of pressure, a bloody, purulent discharge was obtained from the left nostril.

Comment.—The rapid application of pressure induced hemorrhage into the sinus.

CASE 5.—The complaint was that of earache. Examination of the left ear revealed inflammatory changes involving the tympanic membrane. A positive pressure of 4 pounds (203 mm. of mercury) was applied slowly in the compression chamber and was accompanied with a dull aching pain in the left ear. After release of pressure, the patient stated that "something busted in the left ear and my throat filled up with something warm, like fluid that made me swallow. This was accompanied with a whistling sound."

Comment.—Drainage of the middle ear appears to have taken place through the auditory tube.

CASE 6.—In a case of draining otitis media following paracentesis, pressure was raised to 5 pounds per square inch (253 mm. of mercury). On the release of pressure, drainage occurred through the incision.

These outline statements present the principle of securing effective drainage without trauma. The value of the principle of securing aeration and drainage by the employment of a chamber is being evaluated by such studies as Dr. Ivy and his co-workers [14] have been conducting over a period of several years.

ehnke, A. R.: Compressed Air Therapy in to the Sinuses and the Ear, 1932, unpublished

14. Butler, D. B.; Greenwood, G. J., and Ivy, A. C.: Reduced Atmospheric Pressure in the Treatment of Paranasal Sinusitis, to be published.

POSSIBILITY OF DISSEMINATION OF INFECTIOUS MATERIAL BY AIR

Complications involving the ear and sinuses appear to be no more prevalent after pressure trauma, if immersion of the head in water is avoided, than they are in persons not subjected to compression. A factor limiting complications may be the difficulty experienced in equalizing pressure, when even mild degrees of nasopharyngeal infection are present. The thoroughness, however, of aeration of the aural and sinal spaces in diving and aviation gives ample opportunity for the dissemination of infectious material. It would appear then that such modes of infection must be unusual.

CONCLUSIONS

At the present time, the experience gained by physicians working in low pressure chambers has given, and the extensive development of aviation requires, a knowledge of the usefulness and the limitations of the pressure chamber. The obvious value of a pressure chamber lies in its usefulness for the purpose of diagnosis. therapeutic value appears to be in press an adjunct to other forms of treatment a a means of securing nontraumatic and ef ventilation and drainage. The curative va restricted solely to the efficacy of ventilatio drainage in the therapeusis of sinal and disease.

With reference to the traumatic effect duced by differences in pressure, prime cons tions are: (1) the frequent appearance of a ently extensive injury to the structure o middle ear; (2) the spontaneous resoluti these changes without treatment, and (3 absence of complications.

It is on the basis of these considerations specially adapted pressure chambers may b ployed by the otolaryngologist with safety only as an aid in diagnosis but to promote lation and drainage of aural and sinal spac

Lieutenant Commander W. A. White Jr. U.S.N., and Lieutenant Robert Hayter (MC), U obtained the data shown in the illustrations.

STRUCTURE OF THE EXTERNAL NOSE

A STUDY FROM THE POINT OF VIEW OF PLASTIC SURGERY

BRUNO L. GRIESMAN, M.D.

NEW YORK

e final cosmetic result of a rhinoplasty de- not only on the surgical procedures but e healing factors of the nasal tissues of tient. Contractions caused by scars formed the wounds in the layers of the different s (bone, cartilage, muscle, connective tis- leave their mark on the final configuration e nose.

e cannot consider the facial skeleton as a , rigid and dead formation. Like all other of the skeleton, it is constantly subject to tural alterations during the lifetime under fluences of intrinsic and extrinsic stresses. e changes in form and function are greater e early years and less in the later years of "These changing conditions constitute the ic factor of variability in consequence of no two living organisms are ever identical m, structure or function" (Kirk[1]).

e plasticity of the facial skeleton has been oned by Wetzel,[2] who described the effects rtain bites on the nasal framework. He ved a sinking in of the nasal bridge and even evelopment of a saddle nose.

hat is the cause of these forces which influ- the cosmetic result postoperatively? To n answer to this question, I have under- this investigation.

e external nose—i. e., the part projecting e face—is an organic unity from a mechan- tandpoint. The upper and the lower lateral ages seem to be designed especially to resist arrowing of the nasal cavity. Their tech- construction is such that they act in the er of a resilient spring which always re- s its original shape surrounding the vestibu- nasi. When released after being pressed against the septum for any length of time, they open the closed nostrils again.

The tension which exists in the cartilaginous vault can best be compared with the tension in a tracheal cartilaginous arch. Here and there the arch of cartilage is constructed to secure an unobstructed ingress and egress of air. It seems to be evident that in order to regulate the inspiratory and expiratory air pressure by muscular action a cartilaginous spring can be easily distorted. From these considerations one can understand why the distal vault of the external nose could not be made of rigid bone and has to remain cartilaginous during one's lifetime.

The bony nasal arch is built by the frontal processes of the maxilla and by the nasal bones. It is known that the maxilla possesses an extraordinary great firmness. One of the principal forces which exert their influence on the maxilla is the force of mastication. This force is relatively great and produces a great intrinsic stress on the maxillary bone. "A surprisingly great pressure can be exerted by the teeth. In chewing the incisors ordinarily come together with a force equal to 30 or 80 pounds while the molars exert pressure of 140 to 160 pounds in ordinary mastication" (Wiggers[3]).

It has been known from observation of the skin that operative disfigurement will be minimal if the incision is made along the so-called Langer's lines. Langer[4] studied the effects of tension by making holes in the skin of cadavers and noting the direction in which these holes were pulled when the skin was stretched. From these observations he mapped out various planes of cutaneous tension.

Benninghoff[5] studied the functional structure of bone by piercing small holes into the bone of calcified and decalcified skulls with an awl. He demonstrated that after the awl is pulled out the

om the Department of Otolaryngology, Presby- Hospital, and the Vanderbilt Clinic, Columbia rsity College of Physicians and Surgeons.

ad at the meeting of the American Otorhinologic y for the Advancement of Plastic and Recon- ive Surgery, in New York, Nov. 10, 1944.

1. Kirk, E. C.: A Study of the Dynamics Involved Evolution of the Human Dentures in the Rela- f Centric Occlusion, Dent. Cosmos **72**:631, 1930.

2. Wetzel, G., in discussion on Bluntschli: Die hlichen Kieferwerkzeuge in verschiedenen Alters- den, Verhandl. d. anat. Gesellsch. **35**:163, 1926.

3. Wiggers, C. J.: Physiology in Health and Disease, ed. 4, Philadelphia, Lea & Febiger, 1944, p. 771.

4. Langer, C.: Ueber die Spaltbarkeit der Cutis, Wchnbl. d. k. k. Gesellsch. d. Aerzte in Wien, 1861, p. 157.

5. Benninghoff: Spaltlinien am Knochen, eine Methode zur Ermittlung der Architektur platter Knochen, Verhandl. d. anat. Gesellsch. **34**:189, 1925.

tissue breaks into fissures because of tension. In order to make these small splits easily visible, water color, oil color or india ink is rubbed into them. In most cases these holes settle in the form of lines, the so-called lines of tension. These lines show the direction of the effects of stress of the superficial tissue elements, i. e., the

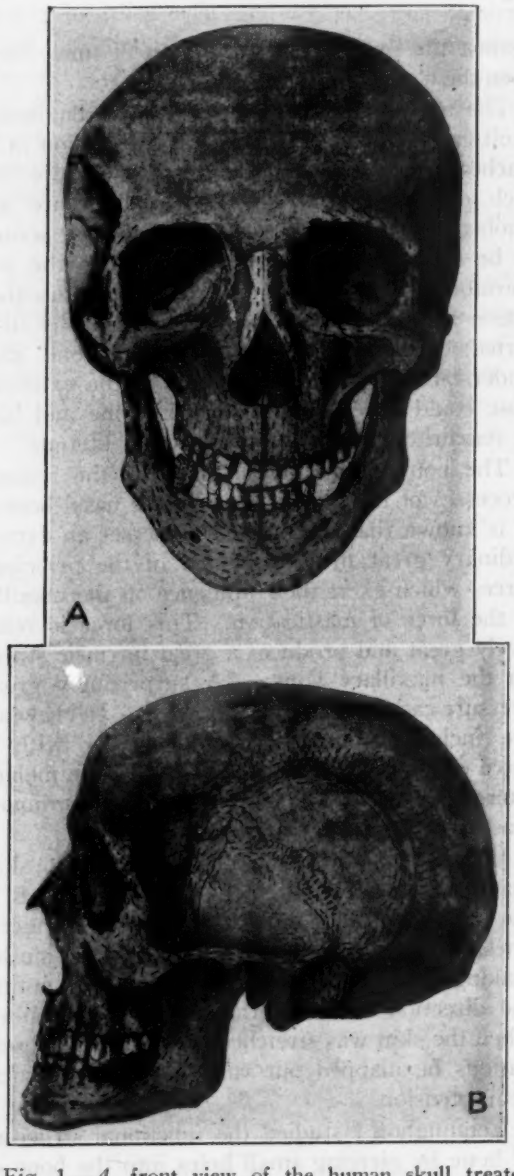


Fig. 1.—*A*, front view of the human skull treated with the split method (Benninghoff [5]). *B*, side view of the human skull treated with the same method.

resultant direction in which the forces of traction and pressure are especially great.

The structure of the bone of the maxillofacial region can be illustrated by Benninghoff's lines.

The nasal or ascending process of the superior maxilla is a triangular plate of bone which projects upward joining the fontal bone, and slightly inward to articulate with the l borders of the nasal bone. The two nasal articulate with each other in tent fashion to the bridge of the nose. Above they are m and strong; below they become thinner wider. The upper borders of the nasal articulate with the frontal bone and its spine. The form and dimensions of the mainly depend on the development of the f processes and the manner in which the bones are set on them (Fomon [6]).

The lines of tension (fig. 1 *A*) which ris the incisor teeth come to an end at the gl as if cut off. Here and at the supra margin a regular arrangement of the li lacking. From a side view (fig. 1 *B*) one c the strong binding of the malar-zygomatic pound. The entire region of the masti influence is clearly visible.

Figure 2 *A* shows a skull with Benning lines. From this picture it is evident th

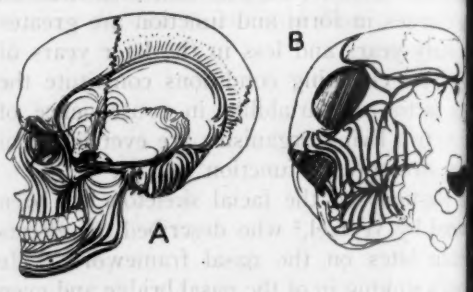


Fig. 2.—*A*, human skull with Benninghoff's *B*, facial skeleton viewed obliquely from below, the basket-like distribution of the lines of stres trayed.

facial skeleton is chiefly constructed in vi the stress of chewing. The lines of stress d halt at the cranial sutures but cross ove edges of the facial bones as if these wer present (Benninghoff [7]).

It may be noticed that the cranium sho dissimilar structure. There is no regula sign. Benninghoff expressed the belief th cranium is thick enough so that no special struction is required by the force of mastic The firmness of the vaulted frontal bone is cient to resist the stress of chewing.

When one looks at the skull obliquely below (fig. 2 *B*), one can observe that the facial skeleton is inclosed all around by which extend from the palatine suture acro alveolar process through the buttresses int

6. Fomon, S.: The Surgery of Injury and R Repair, Baltimore, Williams & Wilkins Company,

7. Benninghoff, A.: Architektur der Kiefe ihrer Weichteilbedeckung, Paradentium **6**:50, 193

of the skull. The facial skeleton is firmly
d with the brain case in a basket-like
er.

one had to fasten the superior maxilla with
s to the brain case, it could not be done
than nature has solved this problem.

figure 2 *A* certain regions of the cranial
are left white. These parts show no dis-
directed lines of stress. Therefore these
s are called neutral zones. In these neu-
ones the lines of stress run criss-cross in
rections.

nninghoff demonstrated that the compact
ancellous parts of the bone have always the
regular or irregular structure. Both tissue
nts together present an inseparable func-
unit.

mparative anatomy gives the answer to the
ion why the lines of stress of the human
skeleton stop at the glabella and at the
orbital ridges.

anthropoid apes the stress of chewing is
directed vertically from the molar teeth up
supraorbital ridges but is carried upward
nally to the skull vault.

ooton [8] described the functions of the brow
s as twofold: In primates whose frontal
ral lobes do not rise above the orbits, the
orbital ridges

protection to the eyes by forming penthouses
them. They also serve as buttresses to resist
remendous upward stress exerted by the jaws in
ng. When, however, the jaws recede and the
l bone rises in a curve above the orbits, the
ridges lose their function. Since the jaws are
nger large and projecting, the upward stress of
ng is diminished and is resisted with perfect effi-
by the high vaulting of the forehead.

enckel [9] examined the skulls of lower and
r animals with Benninghoff's method. He
not find any principal difference in the
of stress among the facial and palatal skele-
of man, the chimpanzee and the orang-utan.
rtheless, there is one remarkable peculiarity
e skull of the goat. This animal is without
r and canine teeth. Therefore there are no
of stress extending from these regions up-
to the orbit. However, lines of stress could
own running from the molar teeth far back-
to the occiput. This seems to be an in-
tive proof of how far the influence of the
s of chewing is exerted on the brain case.

A high factor of safety protects the facial skeleton against forces from other origins (mimetic muscles, traction of soft parts).

Wetzel and Schröder [10] could show that the facial skeleton can withstand stresses nearly eighty to ninety times the average force of mastication.

The form of the superior maxilla determines the profile of the human face. The superior maxilla supports all the upper teeth. The floor of the nostrils, the alae and the base of the columella are fixed by their attachments to the maxilla.

The superior maxilla, because it is part of the upper jaw and because of its neighbors or adjacent important organs, requires a complex con-

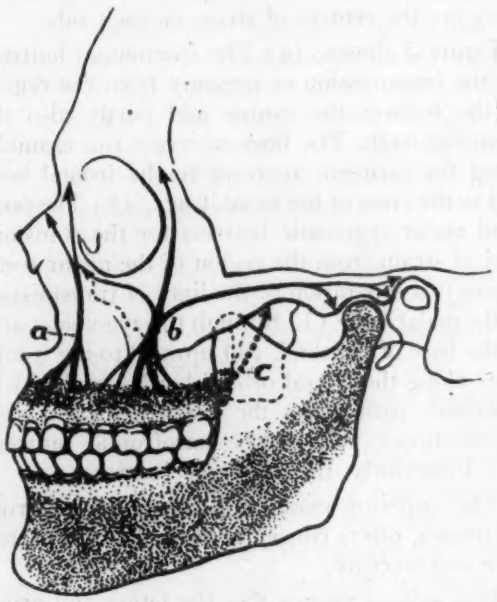


Fig. 3.—The basal arches of the maxilla and the mandible are shown (dotted), and also the buttresses of the facial skeleton: (*a*) the nasofrontal buttress, (*b*) the malar-zygomatic buttress and (*c*) the pterygoid buttress (Bluntschli and Winkler [11]).

struction. Its stability is complicated by the fact that it has to pass around the nasal cavity with the accessory sinuses, especially the maxillary sinus, and to enclose the orbit with the organs contained therein. It is supported by strong buttresses, which can be recognized by visible thickening of the bone at the typical places. The buttresses are connected by means of thin bony plates. These braces stiffen the facial frame.

The superior maxilla shows a narrowing in the region of the roots of the teeth, the so-called

Hooton, E. A.: Up from the Ape, New York, Macmillan Company, 1931, p. 191.

Henckel, K. O.: Vergleichend-anatomische Un-
chungen über die Struktur der Knochenkompakta
der Spaltlinienmethode, Morphol. Jahrb. (Teil 1)
2, 1931.

10. Wetzel, G., and Schröder, B.: Der Sicherheitsgrad im Bau des Gesichtsgerüstes gegenüber dem Kaudruck, Arch. f. Entwcklngsmechn. d. Organ. **105**:120, 1925.

"basal arch" (Bluntschli and Winkler [11]). The maxilla has the least circumference in the region just above the alveolar process. The exerted pressure is transmitted to the superior maxilla through the upper teeth. However, the actual apparatus for the reception of the pressure is the "basal arch" (fig. 3).

Because the force of mastication is exerted from the tooth-bearing segment of the superior maxilla, the buttresses run through the upper component of the bone cranially and have their foundation in the base of the skull, especially in the frontal bone, the root of the nose and the body of the sphenoid bone.

It is these buttresses which in their entirety bear the main pressure of the masticatory effort. The greatest strain is exhibited in their direction. They are the centers of stress on each side.

Figure 3 shows: (*a*) The frontonasal buttress for the transmission of pressure from the region of the incisor, the canine and partly also the premolar teeth. The lines of stress run cranially along the piriform aperture to the frontal bone and to the crest of the nasal bones. (*b*) The powerful malar-zygomatic buttress for the transmission of strain from the region of the molar teeth. There is a separation of the lines of transmission in the malar bone (1) through the zygomatic arch to the base of the skull, (2) upward to the frontal bone along the lateral orbital border, and (3) by a weaker path along the lower orbital margin to the upper part of the nasofrontal buttress. (*c*) Posteriorly, the pterygoid buttress.

The superior maxilla, because of its strong buttresses, offers considerable resistance to pressure and fracture.

It is safe to assume that the biting and grinding force of mastication has a formative influence on the facial skeleton. The force of mastication has a widening effect on the facial framework (Popa [12]).

The superior maxilla does not rise in a straight line; it extends upward in concave and convex curves similar to the vertebral column (Wetzel [13]). This arrangement insures its flexibility and assists in withstanding the stress of longitudinal compression.

11. Bluntschli and Winkler, R.: Kaubewegungen und Bissenbildung, in Bethe, A.; von Bergmann, G., and others: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1927, vol. 3, p. 295.

12. Popa, G. T.: Structure fonctionelle de la duremère crânienne, avec considérations générales sur les facteurs mécaniques crâniens chez les vertébrés en général et chez l'homme en particulier, Ann. sc. de l'Univ. de Jassy **13**:119, 1926.

13. Wetzel, G.: Studien zur Schädelstatik, Verhandl. d. anat. Gesellsch. **31**:216, 1922.

The anatomic construction of the external has to solve two technical problems: (*a*) It buttress the nasal framework and (*b*) it keep the nostrils open.

According to the investigations of Ilberg lines of tension of the cartilaginous vault an subject to the force of mastication (fig. and *B*).

The mosaic-like distribution of the te lines in the nasal septum (fig. 4 *C*) shows precisely the structure of living tissue c sponds with its mechanical function.

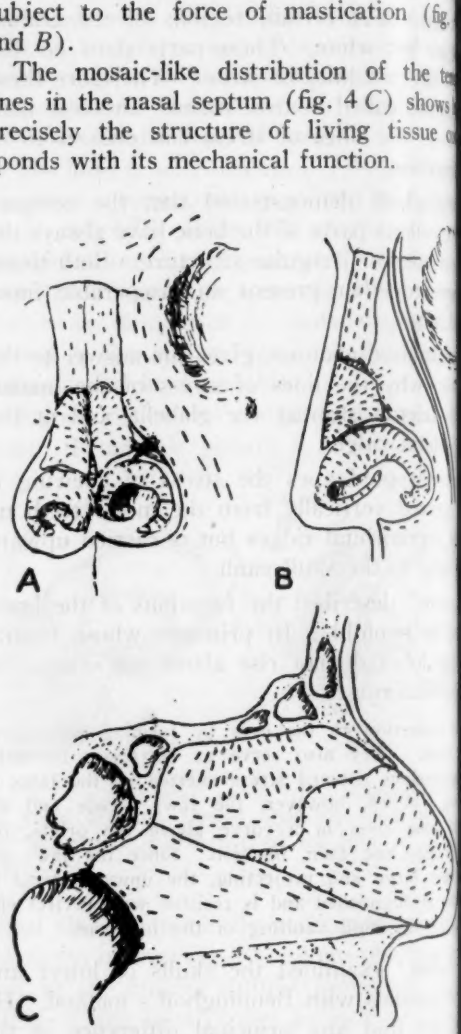


Fig. 4.—The lines of stress of the bony and tilaginous vault of the external nose are shown front view, and *B*, side view (Ilberg [14]). The lin stress of the nasal septum are seen in *C* (Ilberg [14]).

The functional structure of the nasal sep (fig. 4 *C*) is complex and has not yet been quately comprehended.

Ilberg,[14] who first described the lines of ten of the septal mucosa and its bony and cart inous parts, could not give any satisfac explanation.

The perpendicular plate of the ethmoid b is the ossified upper component of an origin

14. Ilberg, A.: Ueber die funktionelle Archit der Nasenknorpel und ihrer knöchernen Umge beim Menschen, Ztschr. f. Laryng., Rhin. & Otol. 239, 1935.

aginous structure. Genetically the vomer is etely different from the other parts of the septum. It is a membrane bone which ops directly from mesenchyma without an ediate cartilaginous stage. All the other parts of the septum originate from cartilag- tissue.

e tension lines of the vomer run obliquely the arch of the hard palate to the body of phenoid bone. It seems probable that they mit the force of mastication.

e cranial part of the perpendicular plate orts the lamina cribrosa. Therefore the ten- lines of the plate extend almost vertically.

e cartilaginous septum shows a functional ture according to the different action of the and the lower cartilaginous vault.

e upper lateral cartilages can be moved only inward and an outward direction (Gries- s). The less firmly attached lower lateral ages can be moved in several different direc- : 1. Their cranial or caudal displacement s the nares. 2. The nostrils can be dilated narrowed in a transverse line. 3. There is a potentiality to distort them in a ventro- l direction. The tip of the nose can be oximated to the upper lip in a sagittal line. same effect is accomplished by the constrict- muscular force of the pars transversa of the ulus nasalis during the production of certain ds.

e lines of tension of the cranial part of the l cartilage in the region of the attachment e upper lateral cartilages extend obliquely continue into the tension lines of these lages across the bridge of the nose. The tional structure of both archly fused tissues onstrates the stress of the springlike mecha- .

here is a small gap between the caudal bor- of the upper lateral cartilages where the laginous septum forms a part of the bridge e nose (fig. 4 *A*).

the caudal region of the septal cartilage, in the region of the lower lateral cartilages, lines of tension extend in the form of a con- curve. When the nasal tip is pressed sagit- against the upper lip, the elastic recoil of the rbolic trajectory of the septal cartilage coun- cts this movement by restoring the natural tion.

his description presents evidence that the l septum is adapted to different functions that a maximum of effect is accomplished a minimum of material.

In a typical rhinoplasty the bony vault is surgically attacked in order to straighten the crooked nose, to narrow the broad nose and to flatten the hump nose. If a hump is present, it is resected by a horizontal cut through the nasal bones with a nasal saw. To narrow the nose, the nasal processes of the superior maxilla are cut with a nasal saw.

The question may arise whether the typical saw cuts interfere with Benninghoff's lines of tension and with the transmission of force through the buttresses.

Frequently after complete mobilization of the nasal bones and successful narrowing of the osseous vault the narrowed nasal bones show a resilient tendency not to stay in the corrected position but to return to their former place.

Two procedures are necessary to narrow the osseous vault: (1) The newly formed ventral borders of the nasal bones near the septum after the resection of the hump should be approximated as much as possible; (2) after the lateral osteotomy the mobilized nasal bones have to be moved medially according to the desired cosmetic result.

The ventral saw cuts run nearly parallel to Benninghoff's lines of force, but the dorsal cuts cross these lines and split the nasofrontal buttress in its cranial course to the frontal vault.

The defect of the nasal bones after resection of the hump has an elliptic form. The saw cut lines are not parallel but oval. This together with the bony web at the radix nasi makes it difficult to approximate the ventral edges of the nasal bones. The dorsal cuts through the frontal processes of the maxilla show a tendency to return to their former attachment. This is partly due to the formation of callus. Sometimes the periosteum takes an active part in the building of callus and becomes vascularized and thickened. Occasionally the swelling spoils the cosmetic effect for many months, until it is absorbed.

There is little doubt that the new bone should form in a manner best suited to bear the strains of the force of mastication. The new bone has its trabeculae laid down in the direction of the applied force. This principle is called "Wolff's law." On the other hand, it is known that excessive irritation or movements of the fractured ends form an undue quantity of callus. Even a moderate amount of play between the fractured edges promotes union and callus formation. Therefore chewing of hard food may help to throw out more callus and effect a more rapid union, which the plastic surgeon intends to avoid in the interest of a good cosmetic result.

Griesman, B.: Muscles and Cartilages of the from the Standpoint of a Typical Rhinoplasty, . Otolaryng. **39**:334 (April) 1944.

Of equal importance is the fibrosis which takes place in the surrounding soft parts.

It is a routine method to make the periosteal separation just wide enough to admit the saws, since extensive stripping may result in necrosis of the bones. The tension lines of the perichondrium and the periosteum (Ilberg [14]) demonstrate (fig. 5) that there is a definite stress in the periosteal layer which may help to draw the adherent nasal bones into their original position. As shown in the picture, the tension lines run in almost the same direction as the fibers of the muscles, intersecting the bony lines of tension.

From the standpoint of a good cosmetic outcome the plastic surgeon is not interested in a quick solid bony union along the line of the lateral osteotomy.

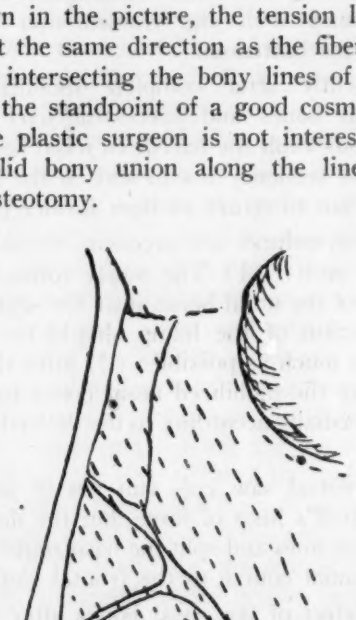


Fig. 5.—The lines of stress of the periosteum and the perichondrium of the external nose are shown (Ilberg [14]).

It is a fact that fractures of the maxilla are united soon by connective tissue, but they need a relatively long time to become united by new bone. The average time may be about seven to nine weeks. Any act of shortening the bony reunion has an unfavorable cosmetic effect on the narrowed nasal bridge. It can be assumed that besides the strain of the mimetic muscles on the facial bones the force of mastication represents the strongest physiologic stress on the facial skeleton and its soft parts. Therefore the stress of chewing should be avoided during the first week after a rhinoplasty. As long as swelling and hematomas of the nasal tissues exist, strong pressure cannot be exerted anyway. Even after one week the patient should not be permitted to use the incisor and canine teeth to bite off a piece of bread or to chew any hard or t food. The nutriment should consist of ch meat and vegetables. There seems to b objection to the use of the lateral teet chewing, especially for chewing vegetabl crush the cellulose membranes. Chewing of food with the front teeth should be avoide at least six weeks.

In cases of plastic alteration of the no which only the cartilaginous vault was ope on, no special restriction of chewing ap to be necessary.

Following a successful narrowing of the sum, a hematoma frequently develops arou eyes. The patient should be informed tha hematoma contains not only essential ma for the reconstruction but also for prote against infection.

It is obviously inappropriate after the co sion of the operation to have the patient his nose by forcing air throught it.

Sawdust, debris, mucous membrane lining periosteal tissue may settle between the fragments and interfere with the narrowing

Sawdust, debris and blood should be care removed with a curet or with a suction

CONCLUSIONS

1. The force of mastication is the main ph logic stress exerted on the facial skeleton vertical and a longitudinal direction.

2. The nasofrontal, malar-zygomatic and p goid buttresses transmit the chewing stres the skull vault.

3. The functional structure of the nasal sep is complex: (*a*) The vomer seems to tra the chewing stress to the body of the sphe bone. (*b*) The perpendicular plate of the moid bone supports the lamina cribrosa. The cranial portion of the septal cartilage the upper lateral cartilages form together a ient mechanism to keep the nostrils open. caudal part of the septal cartilage counte sagittal displacement of the nasal tip.

4. In a typical rhinoplasty the lateral os omy interrupts the transmission of the forc mastication.

5. A quick reunion of the edges of the sa bones after lateral osteotomy must be avo in order to narrow the nasal bridge.

6. For about six weeks after the nasal pl chewing of hard or tough food should be omi Biting off with the incisors should be prohib Meats and vegetables should be chopped mashed.

3 East Sixty-Sixth Street.

OTITIS EXTERNA AND ITS TREATMENT

LIEUTENANT COMMANDER EMANUEL SIMON (MC), U.S.N.R.

The frequency and the severity of otitis externa mine its economic importance. The present rt concerns a group of 90 patients who were rved in a tropical climate and treated until were well. They were all males aged from o 43 and included 76 men of the United s Navy, 2 English sailors, 1 Dutch sailor 3 natives. There were a number of others could not be kept under observation until l. In a period of six months there were over 150 persons with otitis externa in community. In a previous station of roughly this population on the northeastern sea- of the United States I observed about atients with this condition for a period of months. Naturally, the physician and the nts in these two localities view this disease different conceptions of its importance.

he etiology of otitis externa is confusing. extreme attitude is that of Greaves,[1] who e after a survey of 75 cases:

> The etiological agent in the otitis externas is ful. Bacteriological studies, consisting of smear, ng drop and culture, failed to show the presence thogenic fungi in any case. . . . Staphylococci streptococci were extremely rare, but all cases d the presence of a gram negative, motile, pig- producing bacillus of the pseudomonas, or pyo- ous, group.

ws[2] in an excellent study presents a con- ing opinion:

> When the external canal alone is involved the ia are probably secondary invaders but this point ficult to determine in a location where staphylocci constantly present.

er series of 33 cases the condition was due spergillus niger in 14, to Aspergillus fumi- s in 6, and to Aspergillus nidulans in 3. he rest the organism was undetermined.

Williams, Montgomery and Powell[3] stated:

> . . . Thus many otologists would emphasize the factor of fungal infestation in the causation of dermatitis of the external ear whereas according to the dermatologist dermatitis involving the external portions of the ear and the retroauricular region may result from many diverse factors.

They found by special cultural methods that streptococci frequently occurred in otitis externa and retroauricular cutaneous infections and expressed the opinion that these organisms were etiologic.

In a study of 93 cases Minchew, Collins and Harris[4] demonstrated by direct smear that diphtheroids and cocci were present, with no appreciable difference in the infected and the uninfected canal. They failed to find a prevalence of streptococci and thought that this might be explained by the special cultural methods used by Williams and co-workers. In this same group of cases there were 27 in which fungi were present in one or both ears, in all of which Aspergilli were cultured except for a case of infection with Penicillium. Of the cases of infection with Aspergilli they found A. flavus in 10, A. fumigatus in 5, A. niger in 3 and A. sydowi in 4; in 4 cases the species was unidentified. In a comparative study of 22 cases Hatch and Rowe[5] stated:

> . . . A great number of and variety of Staphylococci and Streptococci were always found in the material used for inoculation and consequently in the original swab. It was only by picking the fungi of the higher order that pure growths were obtained and used for detailed study and examination.

Among their cases the otitis externa was recorded as due to A. niger in 12, to A. flavus in 6, to A. albus in 2 and to mixed A. niger and A. flavus in 2.

From these contributions it is apparent that finding an organism in a culture of material taken from an area of otitis externa does not absolutely indicate that the organism is the

his work was done with the help of Navy per- l.

s article has been released for publication by the ion of Publications of the Bureau of Medicine urgery of the United States Navy. The opinions iews set forth are those of the writer and are not construed as reflecting the policies of the Navy rtment.

1. Greaves, F. C.: Phenyl Mercuric Nitrate in the ment of Otitis Externa, U. S. Nav. M. Bull. **24:** Oct.) 1936.

2. Enlows, E. M. A.: Mycotic Diseases of the Ear, nn. District of Columbia **4:**217-222 (Jan.) 1935.

3. Williams, H. L.; Montgomery, H., and Powell, W. N.: Dermatitis of Ear, J. A. M. A. **113:**641 (Aug. 19) 1939.

4. Minchew, B. H.; Collins, B. E., and Harris, M. M.: External Ear Disease with Reference to Fungus Type, South. M. J. **33:**1345 (Dec.) 1940.

5. Hatch, W. K., and Rowe, B.: Fungus Disease of the Ear, Lancet **2:**1561, 1900.

primary invader. It may or may not be the one producing the lesion. It is important to take the material for culture from the infected portion of the canal and throughout the infected area and from the debris when present. The medium used for culture influences the study. Williams and associates [3] pointed out that it is impossible to fulfil Koch's postulates in investigations of dermatids of the skin. If there is sufficient corroborative clinical data, however, one can arrive at some accurate etiologic conceptions.

A basic factor in the causation of otitis externa is a warm or tropical climate. First, it favors the growth of organisms. Second, it increases perspiration, washing and bathing, which afford opportunities for water to get into the external auditory canal more frequently. I am impressed by the increase of otitis externa in persons who get water or perspiration into the external canal. Others have pointed out, and I have noted, that as the weather became warmer the cases were more abundant. It has been mentioned frequently that otitis externa follows swimming; I have seen it often after showers have been taken, and while I have been at this station, I have observed 5 cases in which it followed syringing of ears for wax. Treatment was longer in the few men who were allowed to continue their work near boilers, furnaces or stoves. There was difficulty in keeping their canals dry. I think treatment is prolonged in a tropical climate.

Trauma from rubbing or scratching follows itching. Itching may arise from the irritation due to the presence of a foreign body or perhaps from the irritation caused when the organism begins to invade the tissues. At any rate itching and the subsequent rubbing are the forerunners of the infection. I suggest to persons prone to these attacks that they refrain from rubbing or scratching the canal even if it does itch. Whether the water brings in the organism or the organism is present in the flora of the skin of the canal or is on the rubbing finger is difficult to determine. The canal should be dried gently and well.

McBurney and Searcy [6] in their study of fungicides wrote of molds:

> . . . The relationship of all of these as primary invaders has not been well established, since animal inoculations that have been tried with the isolated fungi have almost invariably yielded negative results.

Enlows [2] tried to produce otomycotic infections and concluded:

> . . . However, all our efforts to produce disease of the external auditory canal failed except when trauma was produced. We were then able to secure g of the fungi in the canal with alterations in the ture of the canal wall not unlike those seen in h ears. Considerable time elapsed from the time of i lation until the first evidences of infection were ob usually 14 to 21 days.

Chisolm and Sutton [7] in a careful discu said:

> In the ear, the fungus is probably never a pr invader but occurs secondary to some other path condition, . . . however, it may persist aft primary condition has disappeared entirely or b masked by it.

In their textbook on Aspergilli, Thom Church [8] wrote:

> . . . There is little doubt as to the occasional f of aspergilli within the external ear, but the rec inoculations in the laboratory leaves a serious do to the primary responsibility of any of them, alt such forms as A. niger are clearly able to ma and probably occasionally extend an ulcerated ar

In the present series of 90 patients ma for culture was taken with a sterile aural s lum and with a sterile curet, which was g scraped over the involved portion of the c and rubbed onto agar slants or dipped into l Material for a second culture was taken both the wall of the canal and the discharg debris in the canal and transplanted onto Sa raud's maltose agar slants. I felt as Mi and co-workers [4] pointed out that Petri d are more easily contaminated in an atmosp laden with spores and organisms. The stan Sabouraud medium used had a p_H of 5.5 Frequently bacteria, particularly Pseudom aeruginosa, would grow in this medium. avoid this difficulty 0.5 cc. of normal phosp acid was added to 5 cc. of Sabouraud's me just after sterilization. This brought the p 3.5 to 4, and bacterial growth was rarely in this medium.

Cultures of material taken from the ea these 90 patients yielded bacteria in all c P. aeruginosa (pyocyanea) in 45, staphylo in 20, Bacillus proteus in 15, Bacillus coli i diphtheroids in 6, and one different orga in each of the remainder. Cultures on Sa raud's maltose agar were found positiv only 19 cases (21 per cent) and were di as follows: 9, A. niger; 5, A. fumigatus Monilia albicans; 1, A. flavus; 1, Asperg tereus; 1, Aspergillus glaucus.

It is apparent that in the majority o cases the otitis was due to bacterial infec

6. McBurney, R., and Searcy, H. B.: Otomycosis, Ann. Otol., Rhin. & Laryng. **45**:988 (Dec.) 1936.

7. Chisolm, J. J., and Sutton, A. C.: Otomy Arch. Otolaryng. **2**:543 (Dec.) 1925.

8. Thom, C., and Church, M. B.: The Aspe ed. 1, Baltimore, Williams & Wilkins Company, p. 79.

is a question as to the primary invader cases in which both a fungus and a bac- organism were observed on culture. It logical that the aspergilli, which are phytes, readily obtain such an environment macerated moist tissue and the discharge the invading organism. They probably in- the superficial layers of the epidermis, not eep layers. In the cases of severe involve- with marked edema and pain or infection a us growth was never obtained on culture. ieve that bacteria are the organisms that e the skin. If this view is correct, an at- must be made to overcome the bacterial ion in all cases; removal of macerated epi- m and debris and drying will do away with condary parasites.

nearly every case it was possible to obtain tory of itching early in the course of the se. Often there was some fulness; later, loss of hearing, depending on the degree struction in the canal and the weighting e drum with edema, discharge or slough. nfection continued, pain appeared in most Frequently there were one or more local- furuncles or there was marked diffuse a extending even into the concha, with hondritis. Preauricular or postauricular a, depending on which wall of the canal involved, was present in the latter type. harge was practically always present. Often s slight and consisted of moist or macerated ficial epithelium, at times called flaky or amating. At times, wax was mixed with discharge, or one saw the well described ceous, soggy mass of debris filling the canal. debris did not always yield a fungus on re. Less frequently one would get a slough the canal wall or the drum. These were o varieties: The first was a moist dead of tissue or a cast of the canal suggestive e presence of a fungus and occasionally ing a black coating, indicating black fruit- odies of a fungus. In these cases there was rness on movement of the auricle with a pain. The second appeared as a fresh e piece of skin and came in the ears with ed edema. This occurrence was usually iated with severe pain. The discharge at accumulated as a dry crust, usually in ateral third of the canal. If one carefully sed the canal thoroughly, there would be d an erythematous or congested skin. This hema disappeared slowly, long after the dis- ge had stopped and the patient had ceased plaining of symptoms. It was noted that ng was usually the first and the last symp- tom. I am reluctant to stop treatment notwithstanding cessation of discharge and disappearance of edema if the patient complains of itching. I have observed recurrences when I thought the patient was well.

Chisolm and Sutton [7] gave the pathogenesis as follows:

> If the fungus grows superficially it may cause no symptoms whatever, or perhaps only a slight itching and later a feeling of fulness and impairment of hearing due to mechanical obstruction of the canal. In such cases, if the patient scratches the ear or tries to clean out the canal, trauma so inflicted promotes penetration to deeper tissues. This may be followed by intense itching, swelling of the canal, tenderness and dull pain.

Dart [9] mentioned the fact that:

> . . . It is possible for the fungus to be present without causing symptoms or producing any pathologic changes; if water enters trouble begins.

Occasionally following prolonged otitis externa the skin becomes dry and papery looking, at times has a slight crust on it, and has been described as branny, lichenoid and eczematoid. This is probably what has been called the dry type of otitis externa. There is doubt as to its pathogenesis. It has been called a dermaphytid, and Sutton and Sutton [10] stated that it is free from demonstrable fungi and depends on hyperergy to products of fungi for its existence. Levine [11] expressed the opinion that it is due to an allergic or sensitive state. Perhaps it is a dermatitis due to a drug or a dermatitis due to discharge from an infective agent. Fortunately, there were only 2 such cases in this group, viz., 14 and 29.

The diagnosis of otitis externa is usually not difficult. The symptoms of itching, with or without pain, the edema and the discharge usually make the diagnosis certain. With marked edema it may be difficult to determine the presence of otitis media or even mastoiditis. In the latter the edema of the posterior wall of the canal is rarely as pronounced as that in severe otitis externa, in which often edema of the anterior wall is present. With gentleness one can insert a narrow speculum and get a view of the drum. Infrequently there may be otitis media subsequent to otitis externa or vice versa; the history will usually indicate the initial infection. With otitis media there are more systemic reactions,

9. Dart, M. O.: Otomycosis: Treatment with Silver Picrate, Arch. Otolaryng. **31**:885 (June) 1940.

10. Sutton, R. L., and Sutton, R. L., Jr.: Mycotic Dermatitis, in Tice, F.: Practice of Medicine, Hagerstown, Md., W. F. Prior Company, Inc., 1932, vol. 4, p. 131.

11. Levine, B.: Use of Phenyl Mercuric Nitrate in Tinea and Yeast Infections of Skin, J. A. M. A. **101**: 2109 (Dec. 30) 1933.

such as a rise of temperature and an increase of white blood cells, and a history of an infection of the upper respiratory tract.

The variety of treatment offered for otitis externa and particularly for that under the name of otomycosis is considerable. McBurney and Searcy [6] in their study of fungicides noted 60 different chemicals used to obtain a cure. They tried 69 substances and combinations to determine the most effective "in vitro" fungicide for aspergilli. They concluded that 2 per cent thymol in cresatin ranked as one of the most effective. They mentioned thorough cleansing of the canal, with use of various recognized antiseptics for different types of inflammation. They made the comment:

> Of the many fungicidal and bactericidal agents used, varying degrees of success have been obtained; that is, their use has allayed and not cleared up the condition; they have required multiple applications, or they have acted as tissue irritants; they have been ideal in the hands of one worker and not with others.

Lederman,[12] who originally advocated cresatin (metacresyl acetate) in otolaryngologic literature, repeated his preference for this drug for furunculosis of the external auditory canal. Whalen [13] has used cresatin and had patients use a 1 per cent solution of thymol in alcohol twice a day for five days in each ear for a five minute period. He also dried the ear with a blast of warm air after cleansing the canal. Stokes [14] cleansed the ear of all debris mechanically and by gentle irrigation with warm water, then medicated the canal with 2 per cent thymol in cresatin. W. D. Gill [15] was an advocate of cresatin and also suggested roentgen irradiation of the infected areas in ears which did not do well after prolonged treatment. K. Gill [16] also believed that cresatin was most effective, producing exfoliation of the top layer of epithelium, allaying pains, preventing sporulation and acting against secondary infection. Chisolm and Sutton [7] were in favor of giving potassium iodide by mouth in courses of two weeks' duration with intervening rest periods of two weeks. Patients were instructed to clean the canal daily and drop in 2 per cent salicylic acid in 50 per cent alcohol. The treatment, applied in 9 cases, usually continued for months.

12. Lederman, M. D.: Cresatin in Laryngology and Otology, Laryngoscope **50**:748 (Aug.) 1940.

13. Whalen, E. J.: Fungous Infections of the External Ear, J. A. M. A. **111**:502 (Aug. 6) 1938.

14. Stokes, H. B.: Fungal Infections of the External Ear, J. Omaha Mid-West Clin. Soc., 1940, p. 60.

15. Gill, W. D.: Otomycosis, Ann. Otol., Rhin. & Laryng. **47**:189 (Oct.) 1938.

16. Gill, K.: Otitis Externa Mycotica, Arch. Otolaryng. **16**:76 (July) 1932.

Schamberg and Kolmer [17] in a study chemotherapy of fungous infections repo all the dyestuffs were "markedly inferio other medicaments." They were also am first to show the relatively poor inhibiti ity of salicylic acid as applied to fungi an tioned that perhaps the exfoliative effect acid rather than its fungicidal influence reason for its effectiveness.

Greaves,[1] after Levine's [11] success with mercuric nitrate in dermatologic infecti to fungi, advocated this drug for otitis e He commented on the fact that fungi metabolic process may produce an ep phytid and that this should be treated little irritation as possible. In the 2 case all medication was stopped, and the ca cleansed thoroughly and dried. The c was helped with 5 per cent coal tar us or at most twice. The tar became irrit used longer in these cases.

Many otologists, as mentioned, list i routine of treatment cleansing of the Hatch and Rowe [5] expressed themselves lows: ". . . but drugs used are not o selves so important as frequent cleaning." tow [18] advised a thorough mechanical c of the canal followed by application of cent salicylic acid in alcohol. Willia co-workers [3] wrote:

> We formed the working hypothesis that the i factor, of whatever nature, had as its pabu detritus in the external auditory canal and that requirement for treatment should be the carefu tion to cleansing the external auditory canal a of any foreign material.

This point was brought out factually by In a comparative study of treatments in cosis he showed that cleansing of the ea cured 51 per cent of 11 patients in h group and 47 per cent of 15 patients see wet season. As mentioned previously, I that the Aspergillaceae are saprophytes an agate on macerated tissue and debris. material is removed and the canal kept dr disappear from the infection. I repeat t primary infection is probably bacterial. I that with recurrences even after months th bacteria and fungi usually were observed cultures. Perhaps they are present in th neous flora of that individual patient spores are present in the superficial epi

17. Schamberg, J. F., and Kolmer, J. A.: therapy of Fungus Infections, Arch. Dermat. **6**:746 (Dec.) 1922.

18. Bristow, W. J.: Otomycosis, Ann. Otol., Laryng. **41**:578 (June) 1932.

19. Reeh, M. J.: Treatment of Otomycosis Otol., Rhin. & Laryng. **51**:146 (March) 1942.

treatment I offer was arrived at after ial and error. For over two years pre- o this work I had been treating otitis with sulfathiazole, 1 Gm. four times times a day, with no local treatment er except for gentle removal of discharge bris. The results were excellent. The , instead of undergoing treatment and sting at times for two weeks and a suc- of furuncles, were able to carry on their tampons of soluble sulfathiazole, and others, but they seemed to be much less effective in this area.

The treatment established in the first 30 cases as shown in table 1, was: (1) thorough cleansing and drying of the external auditory canal and drum membrane every other day; (2) administration of sulfathiazole, 1 Gm. four times a day. This dose was later given every four hours of the day in most cases.

TABLE 1.—*Data on First Thirty Cases*

Organism Obtained on Culture		
Agar	Sabouraud's Medium	Treatment
		Mild Cases
aeruginosa	No growth	Sulfathiazole 1 Gm. q.i.d. 4 days
coli	No growth	Sulfathiazole 1 Gm. q.i.d. 4 days
aeruginosa	No growth	Sulfathiazole 1 Gm. q.i.d. 4 days
aph. aureus	No growth	Sulfathiazole 1 Gm. q.i.d. 10 days, followed by same dose q.4 h. 5 days
aeruginosa	No growth	Sulfathiazole 1 Gm. q.i.d. 4 days
		Moderate Cases
aeruginosa	No growth	Tried 2% salicylic acid 1 week, 1% thymol in cresatin 2 weeks; then sulfathiazole 1 Gm. q.i.d. 10 days, followed by same dose q.4 h. 8 days
aph. aureus	No growth	Sulfathiazole 1 Gm. q.i.d. 5 days
phtheroids	A. niger	Sulfathiazole 1 Gm. q.i.d. 5 days
proteus	No growth	Sulfathiazole 1 Gm. q.i.d. 7 days
aeruginosa	No growth	Sulfathiazole 1 Gm. q.i.d. 7 days; "fungus" in ear 3 months ago
aeruginosa	A. fumigatus	Sulfathiazole 1 Gm. q.i.d. 7 days; similar attack 2 mo. ago and one 5 mo. ago
proteus	No growth	Sulfathiazole 1 Gm. q.i.d. 5 days, then q.4 h. 9 days
aph. aureus	No growth	Sulfathiazole 1 Gm. q.i.d. 5 days; fungus in ear 3 mo. ago; "heat treatment" to ear for 11 days just before arriving here
proteus	No growth	Used 2% salicylic acid 1 week, 1% thymol in cresatin 9 days. Sulfathiazole 1 Gm. q.i.d. 12 days; still a slight discharge; 5% coal tar used; discharge disappeared after 2 days
aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 5 days
aeruginosa	A. niger	Sulfathiazole, 1 Gm. q.i.d. 2 days, q.4 h. 4 days
icrococcus catarrhalis	No growth	Sulfathiazole 1 Gm. q.4 h. 11 days
aph. aureus	Monilia albicans	Sulfathiazole 1 Gm. q.4 h. 11 days
aph. aureus	A. niger	Sulfathiazole 1 Gm. q.4 h. 8 days
aph. aureus	A. niger	Sulfathiazole 1 Gm. q.4 h. 4 days
phtheriods	No growth	Sulfathiazole 1 Gm. q.4 h. 3 days
aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 4 days
iphtheroids	No growth	Sulfathiazole 1 Gm. q.4 h. 5 days
aph. aureus	A. fumigatus	Sulfathiazole 1 Gm. q.4 h. 10 days
coli	A. niger	Sulfathiazole 1 Gm. q.4 h. 5 days
		Severe Cases
proteus	No growth	Sulfathiazole 1 Gm. q.4 h. 20 days; right canal was closed by edema
aph. aureus	No growth	Sulfathiazole 1 Gm. q.4 h. 7 days; sulfathiazole level in blood is 4.06 mg. in 100 cc. Dose increased to 1.5 Gm. q.4 h. After 2 days canal still continues to discharge slightly. Dose increased to 2 Gm. q.4 h. for 2 days; level is 7.5 mg. Discharge stopped, but returned 8 days later with yellow crust. Sulfathiazole 1 Gm. q.4 h.; rash developed. Sodium sulfathiazole packs and sulfanilamide granules used and condition cleared up
aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 12 days; canal markedly edematous
coli	No growth	Sulfathiazole 1 Gm. q.i.d. 6 days, q.4 h. 6 days; blood level is 5.7 mg. After 7 more days of 1 Gm. q.4 h. level is 5 mg. Increased dose to 1.5 Gm. q.4 h.; the level is 5.77 mg. Finally increased dose to 2 Gm. q.4 h. for 11 doses; level is 7.9 mg. Trifle of discharge still present after 27 days. Coal tar 5% applied after cleansing canal: discharge stopped permanently
aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 7 days; dermatitis developed; discharge is practically stopped; some sulfanilamide granules blown in, and discharge stopped

pain disappeared in about forty-eight and there were no recurrent crops of es.

y first attempts to treat otitis externa climate I did not think sulfathiazole would much value, being impressed with the that in the tropics these conditions were due to fungi. Sulfonamide compounds een of little or no value in the treatment gous infections of the skin. I tried the which gave fairly good results in the such as thymol in cresatin, salicylic acid,

In cleaning the external auditory canal a curet was used to remove gently but thoroughly all moist macerated epithelium and discharge from the canal wall and drum surface. At times a cotton fluffed applicator was used to rub off the coating of desquamating epithelium and discharge from the drum. When involved, the drum is thickened and not very sensitive. Occasionally the canal was syringed out if matted crust or, in the later group, sulfanilamide crystals were adherent to the canal wall. One should be sure to remove the debris on the anterior wall

TABLE 2—*Data on Later Sixty Cases*

Case	Organism Obtained on Culture: Broth	Organism Obtained on Culture: Sabouraud's Medium	Treatment
			Mild Cases
31	B. coli	No growth	Sulfathiazole 1 Gm. q.i.d. 4 days
32	B. coli, Proteus vulgaris	No growth	Sulfathiazole 1 Gm. q.4 h. 4 days
33	P. vulgaris	No growth	Sulfathiazole 1 Gm. q.4 h. 4 days; opposite ear infected 11 mo. ago
34	B. proteus	A. tereus	Sulfathiazole 1 Gm. q.4 h. 7 days
35	P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 4 days; similar infection in opposite ear 6 mo. ag
36	Gaffkya tetragena	No growth	Sulfathiazole 1 Gm. q.4 h. 4 days; opposite ear treated 4 mo. ago with 2% acid for 2 weeks
37	Diphtheroids	No growth	Sulfathiazole 1 Gm. q.4 h. 4 days
38	Staph. aureus	No growth	Sulfathiazole 1 Gm. q.4 h. 4 days
39	B. proteus	No growth	Sulfathiazole 1 Gm. q.4 h. 4 days
40	B. proteus	No growth	Sulfathiazole 1 Gm. q.4 h. 6 days
41	P. aeruginosa	P. aeruginosa	Sulfathiazole 1 Gm. q.i.d. 4 days; similar spell 1 mo. ago
			Moderate Cases
42	P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 9 days, similar spell 1 mo. ago
43	P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 7 days
44	P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.i.d. 2 days, q.4 h. 4 days
45	Staph. aureus	A. niger	Sulfathiazole 1 Gm. q.4 h. 5 days
46	Staph. aureus	No growth	Sulfathiazole 1 Gm. q.i.d. 2 days (history of sulfathiazole dermatitis); few oped; practically no discharge left after 2 days; sulfanilamide sprayed in and discharge ceased
	Staph aureus	No growth	Patient returned in 8 days with discharge; sulfadiazine 1 Gm. q.i.d. 4 days
47	Staph. aureus	A. fumigatus	Sulfathiazole 1 Gm. q.4 h. 5 days
48	P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.i.d. 4 days
49	B. coli, B. subtilis	Monilia albicans	Sensitivity to sulfathiazole; sulfadiazine given q.4 h. 4 days. Patient h 4 similar attacks in past 4 months.
50	B. coli, P. vulgaris	No growth	Sulfathiazole 1 Gm. q.4 h. 4 days
51	B. coli, diphtheroids	No growth	Sulfathiazole 1 Gm. q.i.d. 1 day, q.4 h. 6 days
52	B. aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 6 days
53	B. proteus	A. niger	Sulfathiazole 1 Gm. q.4 h. 8 days; fungus in opposite ear 6 mo. ago
54	B. proteus	No growth	Sulfathiazole 1 Gm. q.4 h. 6 days
55	B. proteus	No growth	Sulfathiazole 1 Gm. q.4 h. 7 days
56	P. aeruginosa	No growth	Sulfathiazole 1 Gm. q 4 h. 5 days
57	Staph. aureus	A. niger	Sulfathiazole 1 Gm. q.4 h. 20 days; this is case 20, 2 mo. later
58	P. aeruginosa	P. aeruginosa	Sulfathiazole 1 Gm. q.i.d. 6 days
59	P. aeruginosa P. vulgaris	No growth	Sulfathiazole 1 Gm. q.4 h. 9 days
60	B. coli, P. vulgaris	No growth	Sulfathiazole 1 Gm. q.4 h. 9 days
61	Staph. aureus	A. fumigatus	Sulfathiazole 1 Gm. q.4 h. 6 days; this is case 24, 4 mo. later
62	Staph. aureus	No growth	Used 1% thymol in cresatin 5 days; heavy discharge present; sulfathiazol q.4 h. 7 days
63	P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 4 days
64	Diphtheroid	No growth	Sulfathiazole 1 Gm. q.i.d. 6 days, q.4 h. 4 days
65	P. aeruginosa	A. niger	Sulfathiazole 1 Gm. q.i.d. 6 days
66	B. coli	A. fumigatus	Sulfathiazole 1 Gm. q.4 h. 2 days, q.i.d. 2 days
67	P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.i.d. 6 days
68	P. aeruginosa	A. glaucus	Sulfathiazole 1 Gm. q.4 h. 7 days; this is case 15, 4 mo. later
69	Aerobacter, aerogenes, P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.i.d. 8 days
70	Staph. aureus	No growth	Sulfathiazole 1 Gm. q.4 h. 8 days
71	B. proteus	No growth	Sulfathiazole 1 Gm. q.4 h. 10 days; this is case 26 seen 3 mo. ago
72	Staph. aureus	No growth	Sulfathiazole 1 Gm. q.i.d. 2 days, q.4 h. 4 days
73	Staph. aureus	No growth	Sulfathiazole 1 Gm. q.4 h. 4 days; patient states he was treated elsewhere
74	P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 14 days; works in foundry and perspires; trea several days after complaints ceased because of moisture in canal
75	P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 7 days; this is case 11, 3½ mo. later
76	Diphtheroid	No growth	Sulfathiazole 1 Gm. q.4 h. 6 days
77	P. aeruginosa	No growth	Sulfathiazole sensitive; sulfadiazine q.4 h. 4 days; similar infection in o 4 mo. ago
78	B. proteus	No growth	Sulfathiazole 1 Gm. q.4 h. 6 days
79	B. proteus	A. flavus	Sulfathiazole 1 Gm. q.4 h. 8 days
			Severe Cases
80	P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 4 days; 3 distinct furuncles visible
81	B. coli	No growth	Sulfathiazole 1 Gm. q.4 h. 6 days; marked edema throughout canal
82	B. proteus	No growth	Sulfathiazole 1 Gm. q.4 h. 6 days; recurrence 1 mo. later (case 71); patient treated 3 mo. later (case 26)
83	P. vulgaris, B. coli	No growth	Sulfathiazole 1 Gm. q.i.d. 6 days; had attack 6 mo. ago in both ears
84	B. coli	No growth	Sulfathiazole 1 Gm. q.i.d. 4 days, q.4 h. 2 days; treated for 2 weeks elsewhere markedly swollen, drum thickened
85	B. coli P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 6 days; opposite ear treated 1 mo. ago (case 41)
86	P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 8 days; canal is closed by preauricular edema
87	Staph. aureus, P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 8 days; opposite ear treated 10 weeks ago
88	B. coli	No growth	Sulfathiazole 1 Gm. q.4 h. 14 days; hemorrhagic vesicles, slough; ear treated ago (case 12)
89	P. aeruginosa	No growth	Sulfathiazole 1 Gm. q.4 h. 8 days; edema of canal marked
90	B. proteus	No growth	Sulfathiazole 1 Gm. q.4 h. 2 days; erythema multiforme developed; had e nodosum from last dose of sulfathiazole; sulfadiazine 1 Gm. q.4 h. There was closure of canal with preauricular swelling

canal near the drum margin; in a tortuous it almost forms a recess. Finally the com- surface was dried meticulously with a cotton cator. This treatment was repeated every days. The patients were advised to avoid g water in their ears under all conditions. sulfathiazole was given in the first cases ses of 1 Gm. four times a day with instruc- to avoid the sun and to have light duty nual labor was being performed. Later was found advisable to give this dose every hours. The first patients were put in the nsary, but in time it was found that they carry on with light duty with practically fficulty. They were frequently told to take m. on going to bed and 1.5 Gm. on rising oid waking in the middle of their sleep in to get the necessary 6 Gm. per day.

save space and avoid description of indi- l cases I have divided all 90 cases into groups. The first group comprised the in which usually otitis externa was mild he patient complained of itching and infre- t pains. The edema of the canal was slight, here was a moist desquamation or a flaky arge on the canal wall and rarely on the n. The second group were cases of a mod- type of otitis externa with the patient laining of pain for a varying period of time ittle or no itching. The canal was nar- l by edema, discharge was increased and mes crusted onto the concha, which might nder. The third group included cases of a e type with complaint of pain sufficient to nt sleeping for one or more nights. The a of the canal occluded or almost occluded pening for varying lengths. Often pre- ular edema was present; at times posterior ular edema was seen; occasionally a pal- lymph node was found in these areas. In group there was frequently sloughing of the epidermis came away in pieces and not the macerated membrane-like slough comes with a mold growing on it.

e treatment followed in the second group cases as shown in table 2 was: (1) thor- cleansing and drying of the external audi- canal and drum membrane; (2) light ing of sulfanilamide crystals onto the canal and drum membrane, producing a very coating of granules on these surfaces; (3) nistration of sulfathiazole, 1 Gm. every hours unless noted otherwise.

e application of sulfonamide compounds he canal wall in the treatment of otitis na is not new. A number of otologists at times used sulfonamide drugs in the form of powder or in an ointment or in soluble solutions on tampons. White[20] observed that both sulfanilamide and sulfathiazole inhibit growth of P. aeruginosa, which he found in 32 of 56 cases of otitis externa. Sulfathiazole did not inhibit growth of fungi on Sabouraud's medium, whereas sulfanilamide did. I think that sulfanilamide sprayed onto the canal wall is absorbed in minute quantities if at all. However, it hinders surface bacteria from multiplying, it alkalinizes the discharge, making it somewhat inhibitory to growth of fungi, and finally and perhaps most important it is hygroscopic, taking up moisture and discharge to limit the growth of organisms in discharge.

From my clinical experience I am not impressed with the various bactericides and fungicides used locally on cutaneous or mucosal infections. The degree to which these agents penetrate into the epidermis is questionable even with wetting agents. I think a blood stream distribution of a bacteriostatic agent such as sulfathiazole is more certain to give good results.

TABLE 3.—*Average Number of Days of Treatment*

No sufanilamide in external canal	5.2 5 cases	6.65 20 cases	14.2 5 cases
Sulfanilamide in external canal	4.45 11 cases	6.63 38 cases	7.45 11 cases

In every patient that I had under my direction the symptoms, the discharge and the edema all had disappeared at the end of treatment. I wish to point out that the pain disappeared in twenty-four to seventy-two hours in every case, including those not included in this report, which suggests a bacterial organism as the invader in all cases.

In the first group are certain cases in which I did not consider the otitis as cured by the routine described. These were cases 14, 27 and 29. In every one of the cases of the second group I considered the otitis cured by the routine, though I had to treat the canal for as long as twenty days in some instances. There seemed to be difficulty in obtaining a blood level of sulfathiazole anywhere near 10 mg. per hundred cubic centimeters. It was found necessary to give patients 1 Gm. every four hours in nearly all cases. The number of days of treatment required is averaged for each type in table 3.

The average number of days of treatment was six and eight-tenths days for all 90 cases and six and four-tenths days for the last 60

20. White, S. H.: Local Treatment of Otitis Externa with Sulfonamides, Bumed News Letter **3**:12 (May) 1944.

cases, in which the treatment which I advocate was given. I feel that this is a good average for a tropical climate if one considers all types of cases.

SUMMARY

In a series of 90 cases of otitis externa in a tropical climate there were only 21 per cent in which cultures yielded a fungus. In each one of these same 90 cases it was possible to obtain a bacterial growth on culture, and it is believed that bacteria are the primary invaders in otitis externa. A treatment is offered which was successful in every one of the last 60 cases:

1. Thorough cleansing and drying of the ternal auditory canal and drum mem
2. Spraying of the canal wall and drum brane with a coating of sulfanilamide
3. Administration of sulfathiazole, 1 Gm. four hours, and if the aural condition improved, 1.5 Gm. every four hours. diazine was used when sulfathiazole not tolerated.

The routine must be kept up until the charge and the edema have disappeared the patient does not notice itching. The av length of treatment for all cases in this in a tropical climate was less than seven

FRACTURE-DISLOCATIONS OF THE CARTILAGINOUS NOSE

ANATOMICOPATHOLOGIC CONSIDERATIONS AND TREATMENT

JACQUES W. MALINIAC, M.D.
NEW YORK

ile the pathology and the treatment of nasal fractures are well established, the of injury to the cartilaginous components nose, being less definite, are often mis-reted. In this paper I shall discuss dis-ons of the cartilaginous nasal structures their relationship to external deformities utline a method of reconstruction based anatomic changes resulting from trauma.

ANATOMIC CONSIDERATIONS

fibrocartilaginous structure of the nose ed on the position and the interrelation-the septum and the lateral cartilages, on e hand, and the bony framework, on the

septal cartilage is solidly fixed in a bony formed by the perpendicular plate and the (fig. 1 *A*). The topography of its inferior is of great surgical significance. The border of the vomer presents a groove of g depth in which the septum is affixed hence a cartilaginous prolongation often s toward the sphenoid bone. The maxil-one and the nasal spine in front of the being flat and slightly oblique, offer a surface for the quadrangular cartilage. erichondrium, together with the perios-forms a fibrous capsule responsible for obility of the septum in this area. Thus wer border of the septal cartilage, well posteriorly in the groove of the vomer, es looser over the maxillary bone and the r nasal spine. This also represents its f active growth; it is at this level that nds the spurs which extend along the ocartilaginous articulation.

infants and young children the septum rly always straight. Only at the age of 7 or 8 do septal deviations and spurs start to appear.

Another key point on the septum is its dorsal border with the upper lateral cartilages which form its lateral expansion. At the junction of this border with the nasal bones, depressions occur due to fracture dislocations of the lower septal

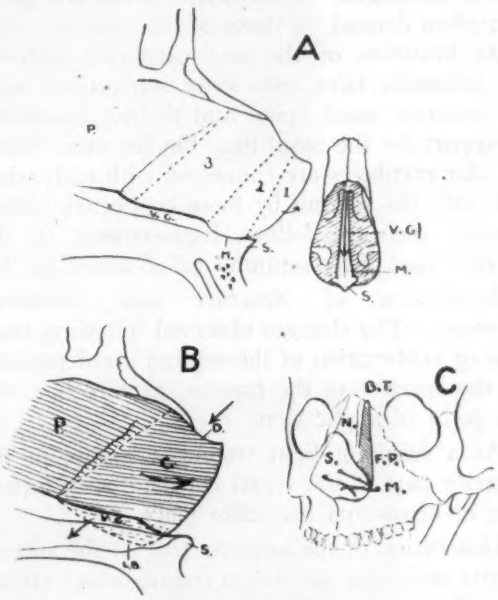


Fig. 1.—*A*, topography of fracture-dislocations of the nasal septum. At the left the anterior segments (*1* and *2*) of the septal cartilage, supported by the sliding surface of the maxillary bone (*M.*), and the nasal spine (*S.*), should be reset in the midline to relieve obstruction anteriorly and to provide support for the nasal tip; they form an angle of deviation with the posterior segment (*3*). *P.* is the perpendicular plate of the ethmoid bone. At the right are the vomer groove (*V.G.*), the maxillary bone (*M.*) and the nasal spine (*S.*) as seen from above.

B, subluxation of the septum on the vomer (*V.G.*) with double deflection of the dorsum. *D.* indicates a dorsal depression caused by traction downward and backward. The arrow below *C.* indicates the lateral displacement of the septum.

C, Joseph's procedure for the correction of a cartilaginous dorsum laterally displaced by traction. *B.T.* indicates a shaded area representing a bony triangle resected from the wider wall in lateral displacement of the bony nose. A silk suture extending from (*S.*) the septum to (*M.*) the maxillary bone was erroneously relied on to correct the deformity by the exercise of prolonged traction on the displaced cartilaginous dorsum. *N.* indicates the nasal bone; *F.P.*, the frontal process.

d at the annual meeting of the American Society tic and Reconstructive Surgery, in New Orleans, 8, 1944.

ne of the surgical procedures described in this are shown in the film "The Fractured Nose—Repair for Deformity and Dislocated Septum" n., silent, sound and colored), registered by the ttee on Medical Motion Pictures, American Col-Surgeons, 1941, no. 457.

border. Such deformities are easily corrected by prompt resetting of the displaced parts after injury (fig. 1 *B*).

The upper lateral cartilages, fused to the dorsal border of the septum, are loosely attached to the lower border of the nasal bones by means of fibrous tissue originating in perichondrium and periosteum. This intimate connection with the septum closely associates them in any displacement of the latter, which is often followed by a tear in the fibrous articulation or by a marginal fracture of the nasal bone, with formation of a depression or a bony callus. (See *A, B* and *C* in figs. 2 and 3.)

The nasal tip and the nostrils form an anatomic entity closely connected with the lower lateral cartilages. Their shape, form and position often depend on those of the septum. The inner branches of the alar cartilages forming the columella have only loose connections with the anterior nasal spine and do not constitute a support for the nasal tip. On the other hand, the alar cartilages are connected with each other and with the septum by loose connective tissue, causing them to follow displacements of the anterior part of the septum [1] (*A, B* and *C* fig. 3).

Mechanism of Fracture and Anatomic Changes.—The changes observed following fracture or subluxation of the septum are dependent on the severity of the trauma, its direction and the point of application.

As a result of light trauma a fissure of the anterior part of the septal cartilage with formation of hematoma may take place.

Dislocation of the anterior part of the septum occurs under the impetus of trauma which strikes laterally at the mobile part of the nose. When the direction of the striking force is anteroposterior and the septal cartilage is caught in the sagittal plane, lesions are at maximum. Then the septal cartilage affixed in the vomer groove posteriorly may bend or fracture laterally in its anterior segment (fig. 1 *A*). Two varieties of fracture of the septum are observed: vertical and horizontal.

The vertical fracture of the septum is the most common (figs. 3 and 4). The line of fracture extends downward and backward from the dorsum toward the nasal spine, dividing the cartilage into two segments: the posterior, which is the larger, and the anterior, displaced in the frontal plane. The dorsal border of the anterior fragment pulls the nasal tip to the same side.

Clinically, a vertical fracture reveals its the oblique position of the anterior segme the septal cartilage inside one nostril. anterior border of the septum, through pre causes displacement of the inner crus o lower part of the alar cartilage, which thu trudes under the vestibular skin.

When the upper lateral cartilage doe resist the impact of trauma, simultaneously the vertical fracture a rupture occurs be nasal bone and cartilage, with or without ginal fracture. The latter often manifests in an exuberant bony callus (fig. 2).

The horizontal fracture of the septum u runs obliquely downward and forward a few millimeters above the free border o vomer. Diagnostically it is often confused subluxation of the septal cartilage from vomer groove, which often occurs simultane with lateral displacement of the dorsum 1 *B*).

If infection does not take place, the ments are unified by fibrous tissue or by a layer of cartilage, as in a greenstick ty fracture. Histologic examinations have s that a post-traumatic bend of septal car occurring in early life often results later ossification and distortion of the cartilage.[2]

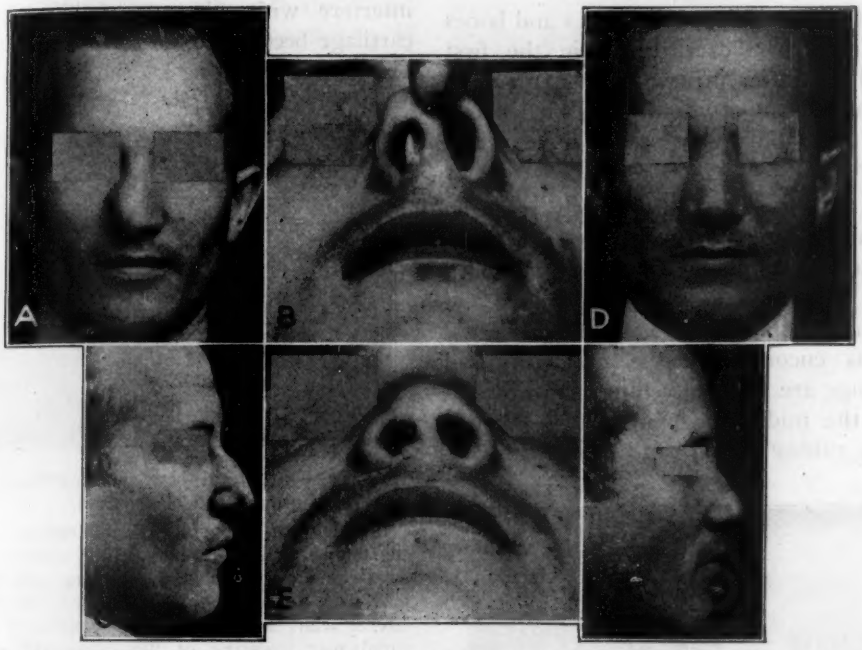
As Mollière [3] demonstrated experimen complete subluxation of the septum on the produces a double deflection of the dorsu depression caused by traction downward backward and a lateral displacement in direction of trauma due to detachment o upper lateral cartilage, often with marginal ture (fig. 1 *B*).

Fracture of the nasal bones frequently ac panies dislocation of the cartilaginous co nents. The nasal bones, which are thick i upper half, thin down conspicuously toward lower border and are intimately united their inner borders, forming a solid bony in the midline. Their thickness varies approximately 7 mm. in the upper half to 0.5 mm. at the lower border. This ex why in lateral trauma a fracture of the bones never occurs in the midline but u usually along the points of least resis namely, at their junction with the frontal esses. In severe injuries, fracture occurs on sides, and there is lateral displacement o entire bony dorsum (figs. 2 and 5).

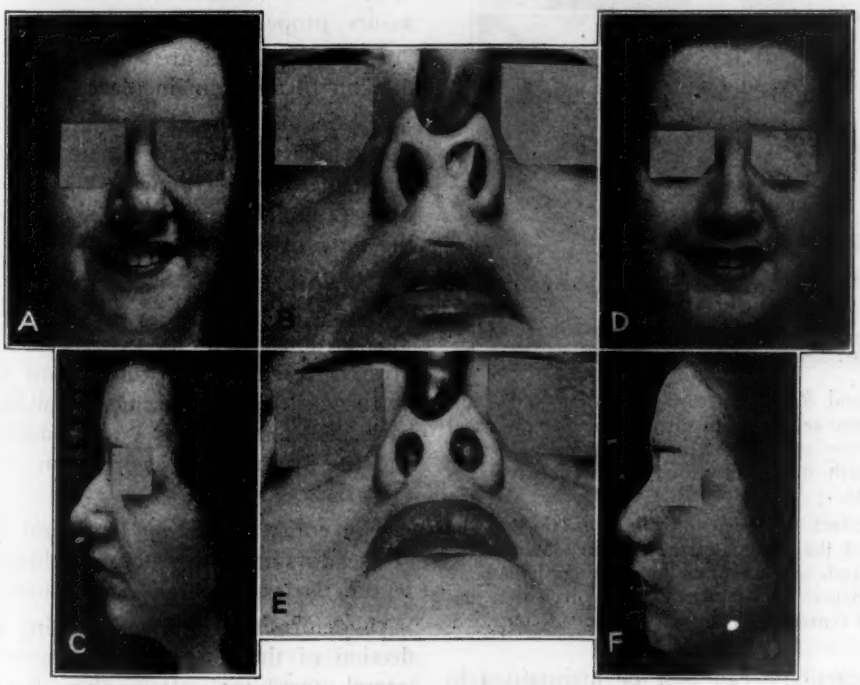
1. Zuckerkandl, E.: Anatomie normale et pathologique des fosses nasales, translated by T. Lichtwitz and P. Garnault, Paris, G. Masson, 1895.

2. Fuchs, K.: Zur pathologischen Histolog inneren Nase, Monatschr. f. Ohrenh. **66**:1077 1932.

3. Mollière, D.: Note sur la chirurgie esthéti nez, Lyon méd. **58**:555, 1888.



g. 2.—*A, B* and *C,* post-traumatic deformity with lateral displacement of the septum and the cartilaginous n, marginal fracture of the lower right nasal bone, and hypertrophy of the right triangular cartilage. *E* and *F,* effect of shortening and resetting of a subluxated septum in the midline with posterior submucous on, lowering of the bridge line, refracturing of the frontal processes of the maxilla and reshaping of the right cartilages. No resection of a bony triangle was required.



. 3.—*A, B* and *C,* fracture-subluxation of the entire septum with double deflection of the dorsum and marginal e of the right nasal bone. The depression along the cartilaginous bridge is caused by dislocation of the ge.

E and *F,* effect of resetting of the deflected septal cartilage in the midline, slight lowering of the bony dorsum, rowing along the frontal processes of the maxilla, and symmetric adjustment of the upper and lower cartilages.

EARLY TREATMENT

The resetting of displaced cartilages and bones should preferably be carried out in the first forty-eight hours after injury. Early treatment is of cardinal preventive importance, particularly in children in whom delay results in developmental abnormalities.

With use of a local anesthetic, early resetting of the septum can be performed even in children. The dislocated quadrangular cartilage is raised and pushed back into the vomerine groove and above the nasal spine. If resistance to digital correction is encountered, the nasal tip and septal cartilage are forcefully lifted forward and pressed to the midline. A periosteal elevator protected by rubber tubing can be used if necessary. The cartilage can best be maintained in place by mattress sutures tied over he dorsum and the columella on a piece of gauze and by endonasal packing.

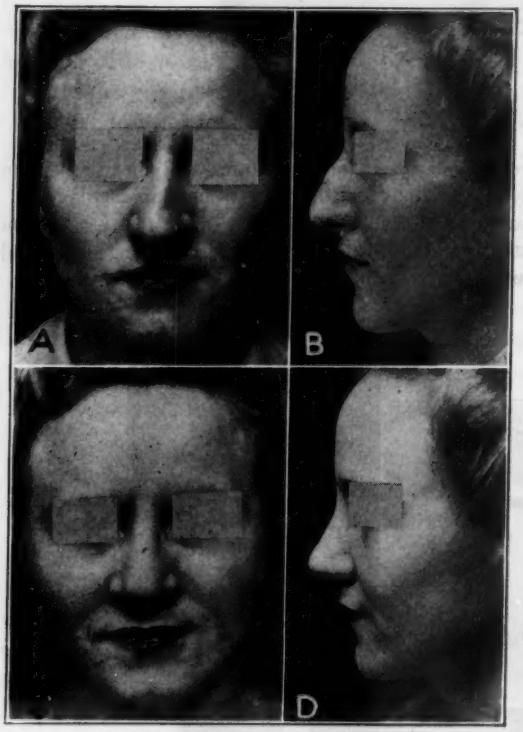


Fig. 4.—*A* and *B*, old fracture-dislocation of the anterior part of the septum with displacement of the nasal tip and the lower segment of the cartilaginous dorsum. There is growth disturbance of the lateral cartilages on the left side.

C and *D*, effect of shortening and resetting of the anterior part of the septum in the midline, lowering of the osteocartilaginous dorsum and narrowing of the bony bridge. The depressed left alar cartilage has been dissected out and reinserted in place, convex side outward.

Where a number of days have elapsed since the injury, resistance may be encountered. After a week to ten days, fibrous tissue formation interfere with adequate mobilization, and cartilage becomes fixed to the side of the w Here open resetting of the septum is req

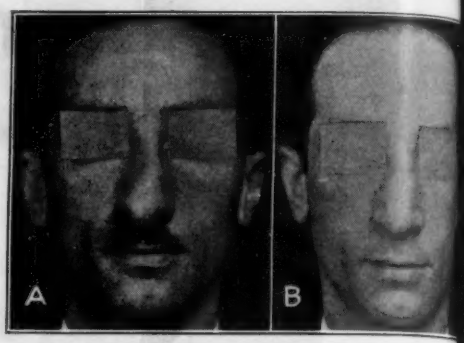


Fig. 5.—*A*, lateral bony and cartilaginous d ment with pronounced obstruction.

B, effect of two stage reconstruction. First s shortening and resetting of the anterior part cartilaginous septum in the midline with posterio mucous resection. Second stage: shortening lateral walls with symmetric adjustment of the cartilages; lowering of the osteocartilaginous d with resection of a bony triangle and infracturing frontal process on the opposite side.

This is done through an incision of the m membrane along the floor of the nose with exposure of the dislocated area. A mat suture carried through the nasal cavity assure proper fixation. (See *4* in fig. 7.

Nasal cartilages are easy to reset but r difficult to maintain in place.

If packing is used as additional suppor the mattress suture, it should be placed i angle of the vault between the lateral wall the septum, above a breathing tube located the floor of the nose.

OLD FRACTURE DISLOCATIONS

It is unnecessary to discuss here the old pressed fracture with downward displace of the cartilaginous frame, resulting in sa back deformity. This is an established e and the manner of its correction is well k (fig. 6).

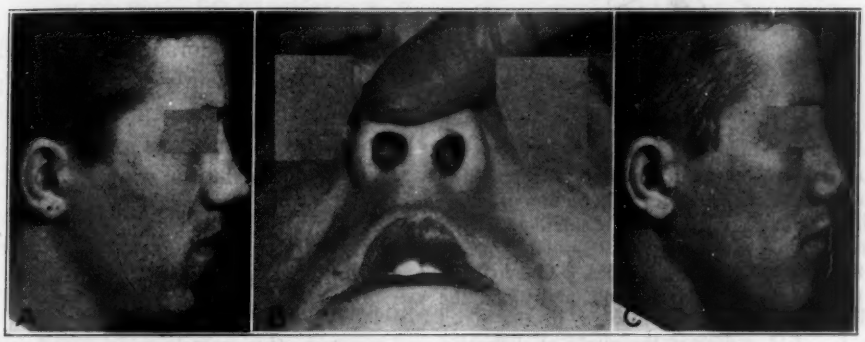
The entity consisting of lateral displace of the dorsum with fracture subluxation o septum is less generally understood.

In deformities of long standing there i flection of the dorsum with asymmetry o lateral cartilages, often plus displacement nasal bones. A concomitant overdevelop of the nose may also be present. When th located septum is primarily responsible fo or part of the external deformity, it shoul dealt with first in order to reestablish sup

e tip and relieve obstruction (fig. 5). Often an be done simultaneously with the correc- of the external deformity (figs. 2 and 3). my experience the procedures which are ently advocated for the repair of this entity proved inadequate (fig. 1 *C*). Joseph's sed on the assumption that the deflected m results from a fracture-deviation of only nterior-superior zone of the septal cartilage, t does not provide for all the post-traumatic es which have occurred. He submits the cartilage to prolonged traction by means heavy silk suture thread attached to the lary bone. If the cartilage is highly re- t, he resects a narrow vertical segment from ptum. In addition, lateral pressure applied by a specially devised compressor is recom- ed to prevent recurrence of the displace-

of a vertical segment of cartilage along the angle of deflection may be required. Incision of the mucous membrane along the floor of the nose on the opposite side is necessary when the lateral traction is pronounced (fig. 7 *3*).

When the angle of deflection is about 90 degrees, the anterior septal fragment rests in the frontal plane across the vestibule and the nasal tip is drawn downward to the side through lack of support. Here a wide exposure of the septal deflection is accomplished by separation of the septum from the columella and undermining of the skin along the lower third of the dorsum. The mucous membrane on the convex side is separated from the anterior border along its entire extent, and incisions are made along the dorsum and the floor of the nose, permitting wide retraction posteriorly. To straighten the angular deflection of the septum, a wedge is re-



g. 6.—*A* and *B*, depressed fracture of the dorsum with pronounced obstruction due to fracture-dislocation e bony and cartilaginous septum.
effect of submucous resection of the obstructing septum; slight lowering and narrowing of the bony bridge ransposition of the triangular cartilages to fill the dorsal depression. (See fig. 8.)

etzenbaum's procedure will release an an- r deflection of the septum with displacement e nasal tip, but it does not reset a deflected laginous dorsum (fig. 7 *2*). Others recom- a submucous resection of the deflected l parts and insertion of a free cartilaginous into the columella to provide support for ip, but this will rarely remedy the dorsal ction.[4]

uthor's Method for Correction of a Deflected ptum with Subluxation.—The exact procedure nds on the extent of fracture and displace- t. When there are angulation and only t displacement of the tip, the septal cartilage cised along its anterior border and along ngulation posteriorly as high as the dorsum ring about complete straightening. Excision

moved along its entire height, but the continuity of the dorsum is preserved. An incision along the lower border of the displaced cartilage permits lateral mobilization. Removal of a strip of cartilage at this level may be required to replace the septum on the vomer, which is preserved (fig. 7 *3a*).

The freed cartilage attached to the mucous membrane on the opposite side can now be freely moved in the horizontal plane. Complete straightening of the dorsum in the sagittal plane, however, cannot be achieved until all the secondary bends are released, either by vertical excisions of narrow segments of cartilage along its entire height or by crisscrossing when curvatures are less pronounced. In all these manipulations it is essential to preserve the mucous membrane on the opposite side.

The resistance which must often still be eliminated before the entire dorsum can be straightened is due to the short and thickened mucous

Peer, L. A.: Management of Deformities of the Ear, Eyelids and Palate in Infants and Small ren, Dis. Eye, Ear, Nose & Throat **2**:166 (June)

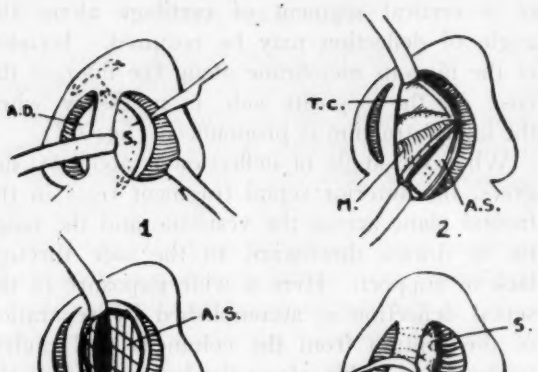


Fig. 7.—Author's procedure for repair of septal dislocations with external deformity: *1.* Correction of angulation and displacement of the septal cartilage. The anterior border of the septum (*S.*) is exposed by separation from the columella on both sides. The incision is continued upward toward the dorsum, and along the upper lateral cartilages, and downward to the nasal spine (arrows). *A.D.* indicates the angle of deflection of the septum.

2. Exposure of the septal cartilage (*A.S.*) beyond the angle of deflection by wide separation of mucoperichondrium (*M.*) extending from the dorsum to the floor of the nose. The mucous membrane on the opposite side is left intact. The removal of a vertical segment of cartilage at the angle of deflection in its entire height is not sufficient to straighten the anterior segment. Its disconnection from the bony floor is required for free mobilization; resiliency of cartilage is eradicated by crisscrossing. The posterior part of the septum beyond the angle of deflection is resected if required. *T.C.* indicates the triangular cartilages detached from the septum and shaped.

3. Correction of a deflection involving the cartilaginous dorsum. The anterior segment (*A.S.*), attached to mucoperichondrium on the opposite side, is incised along the upper and lower borders. A wider exposure is required for straightening the dorsal bend. (See *3a.*)

3a. Formation of a posterior mucoperichondrial flap (*M.*) which facilitates exposure of the cartilage (*C.*) and posterior submucous resection. The anterior segment (*C.*) is mobilized and, if necessary, resected along the floor for resetting in the midline. Crisscrossing of cartilage is done along its entire height. The mucous membrane (*M.*) is left in place. *V.G.* indicates the vomer groove; *S,* the nasal spine.

4. Mucoperichondrial flaps on both sides of the septum (*S.*) brought together anteriorly and posteriorly with mattress sutures.

membrane on the concave side. This is u encountered in old deformities, where g has been impeded for a long period. He anteroposterior incision along the floor o nose is required. The mucous membran on the convex side is now replaced and a by mattress sutures along the entire se (fig. *7 4*). In addition to the mattress su a wired septal splint can be used to immo the septum, making endonasal packing necessary.

If shortening of the septum is indicate is done at this stage. The raw surface o floor of the nose resulting from the relea the shortened mucous membrane may be l enough to require a Thiersch graft dres This will prevent scar formation and recur of traction toward the concave side.

The lateral displacement of the bony b which is frequently associated with deflectio the cartilaginous dorsum is corrected at the s stage or subsequently by the usual procedu releasing the attachment along the frontal p esses of the maxilla and removing a bony tri from the wide lateral wall (fig. 1 *C*).

When the upper lateral cartilages are formed, their symmetry should be reestablish Any fibrous thickening along their insertio the nasal bones is removed (fig. *7 2* and As a rule this cartilage is overdeveloped on convex side and flattened on the opposite s The hypertrophied cartilage is brought d

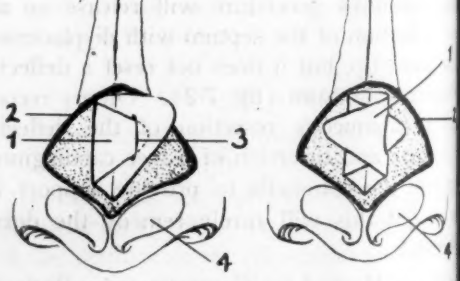


Fig. 8.—Transposition of the upper lateral carti (*1*) to fill out a defect in the middle third of dorsum. The cartilages are separated from the m membrane (*2*) and transposed to the dorsal depres through a split (*3*) made in one of them. (*4*) cartilages.

to the level of the opposite one and is shortened. When one side is conspicuously pressed, transposition of a graft from the hyp trophied cartilage is indicated (fig. 8).

dure, which I described many years ago,[5] proved useful in corrections of dorsal de- ions below the nasal bones (fig. 6).

to the alar cartilages, displacement of the ior part of the septum laterally, with lack pport for the tip, causes downward retrac- of the latter, with flattening or depression e concave side. On the opposite side the berance of the septum, exercising a pro- d pressure from below on the corresponding cartilage, causes deformity of the latter, n remains even after elimination of the pres- (figs. 2, 3 and 5).

mmetry of the tip is reestablished by excis- narrow strip from the hypertrophied car- e and crisscrossing the resilient parts. On pposite side, where the alar cartilage is essed, its reversal or an addition of a graft dicated. A graft from the auricular cartilage st appropriate for this purpose.

the reduction of nasal tip cartilages, it is rtant to avoid the sacrificing of any vestib- skin. Removal of the latter often causes formation and retractions about the tip, h are deforming and difficult to correct. en the septal cartilage is inadequate to pro- support for the nasal tip, a free cartilaginous splant is required. I have not had uniformly good results with preserved cadaveric cartilage inserted anteriorly for support of the nasal tip. The vertical columellar incision and the proximity of the suture line to the graft could be blamed for it. It is usually safer to insert this graft at a stage separate from the general resetting of the cartilaginous components.

5. Maliniac, J. W.: Correction of Nasal Depressions Transposition of Lateral Cartilages: A New Method, Otolaryng. **15**:280 (Feb.) 1932; A Procedure for tion of the Nasal Dorsum by Transplantation of ateral Cartilages, Arch. Otolaryng. **41**:214 (March)

SUMMARY

The position of the septal cartilage in the vomerine groove and its relationship to the other cartilages and to the bony framework constitute the key points of the cartilaginous structure of the nose.

Displacements and fractures of the nasal cartilages in early life should be promptly corrected. Otherwise they interfere with normal growth of the nose and permanent deformity results.

Some of the procedures suggested for the correction of these deformities are inadequate because they fail to take into account the pathologic changes which occur in fracture-dislocations of the nasal cartilages.

The main points of the method of repair recommended are wide exposure of the cartilages involved and reconstruction of all changes in the cartilages themselves as well as in their relationship to the nasal framework. Complete release of all adhesions at the time of surgical repair is essential: No reliance should be placed on postoperative traction.

106 East Seventy-Eighth Street.

TESTS FOR UNILATERAL DEAFNESS

CAPTAIN ROBERT E. PRIEST*
MEDICAL CORPS, ARMY OF THE UNITED STATES

Military otologists must often evaluate claims of total unilateral deafness.[1] Papers on the subject cite many tests for use in this evaluation, but they frequently leave the reader confused as to which are most valuable. While conducting the ear, nose and throat clinic in an army hospital, I have examined many patients who claimed to have total unilateral loss of hearing. I have sometimes suspected that there was total loss of hearing in one ear when the patient did not admit such loss.

Many tests to determine the unilateral loss of hearing have been tentatively used, and a satisfactory examination procedure has finally evolved. It is presented here in the hope that such a routine will be useful to others. The tests described deal only with the diagnosis of severe or total unilateral loss of hearing. Not many patients simulate total binaural loss of hearing.[2] They usually exaggerate existing loss to total unilateral deafness.

The accurate determination of the presence or absence of unilateral deafness in soldiers is manifestly important. Inclusion of deaf men in combat units jeopardizes the units as well as the men so handicapped. Deafened soldiers can often serve well where security does not demand good hearing. The detection of malingerers is essential. Some soldiers deny having auditory loss in order to remain with their units. Others claim deafness to avoid hazardous service.

Men with normal hearing in one ear and total loss of hearing in the other are seriously handicapped. The American Medical Association adopted a method for computing total bin loss of hearing for speech. Persons having loss in one ear are credited with only 12 per binaural loss. This value is assigned when ears have an unimpeded opportunity to Often the ears of soldiers on duty are ma by wind and the noise of machines. A man ing normal binaural hearing can sometimes his head and decrease the masking in one When a man's only hearing ear is masked, of little value as a combat soldier.

Men having unilateral loss of hearing unable to locate sounds accurately. N persons fix the position of sound by turning heads to produce a difference in the aud impressions received from the two ears. these impressions they estimate the locati the source of the sound. This ability i absolute necessity for certain combat sold

Tests to determine unilateral deafness sh be as nearly absolute as possible. Many allow the examiner to suspect malingering. give information but are not definitive. are sometimes useful ancillary procedures. routine for testing patients suspected of ha unilateral deafness should include only the informative tests. The patient's attention and the examiner's time should be conser Tests requiring complex apparatus or presen technical difficulties are undesirable.

Care must be taken to prevent suspe malingerers from becoming wary. If this caution is taken, patients having unilateral ness are unaware that their honesty has assayed. Overconfident malingerers are easily ensnared. Occasionally, when result tests are contradictory, additional inform may be elicited by informing the patient that tests indicate possible dishonesty. When tests are repeated, more conclusive results be obtained. This is especially true with re to patients having unilateral deafness wh audiograms fail to give a shadow curve.[4]

The examining physician is primarily a finder. His proof that hearing exists when

* Division of Otolaryngology, University of Minnesota Hospitals, on leave of absence.

1. (*a*) Harbert, F.: Functional and Simulated Deafness, U. S. Nav. M. Bull. **41**:458-471 (March) 1943. (*b*) Pitman, L. K.: A Device for Detecting Simulated Unilateral Deafness, J. A. M. A. **121**:752-753 (March 6) 1943. (*c*) Weber, H. C.: Simulated Deafness, U. S. Nav. M. Bull. **29**:221-224 (April) 1931. (*d*) Kerrison, P. D.: Diseases of the Ear, ed. 4, Philadelphia, J. B. Lippincott Company, 1930, pp. 94-96 and 553.

2. (*a*) Kerrison.[1d] (*b*) Grove, W. E.: Simulation of Deafness, Ann. Otol., Rhin. & Laryng. **52**:573-580 (Sept.) 1943.

3. Tentative Standard Procedure for Evaluating the Percentage of Useful Hearing Loss in Medicolegal Cases, report of the Council on Physical Therapy, J. A. M. A. **119**:1108-1109 (Aug. 1) 1942.

4. Bunch, C. C.: Clinical Audiometry, St. L C. V. Mosby Company, 1943, p. 56.

nt claims to be deaf is only one item of evi- in the case. This evidence must be exam- in conjunction with the psychiatrist's on and the patient's total personality, cal state and social situation. On dis- ing a malingerer one is tempted to show nation and to take decisive steps toward r punishment. This is not the otologist's ion. He can only do injustice to himself thers by voicing his indignation. When all vidence is collected, punishment can be rly given. If the patient is a psychopath, opriate medical care can be applied.

EXAMINATION ROUTINE

he examination routine finally adopted in- s a careful taking of the patient's history an examination of his ear drums, canals, nasopharynx and pharynx. The distances which whispering and conversational voice be heard are determined. The Rinne and er fork tests are made. Inflation demon- es tubal patency. Results of serologic tests yphilis are obtained. Bone and air conduc- audiograms are made in a sound-insulated . Audiograms and tuning fork and voice are often repeated to demonstrate suspected nsistency. When the patient claims to have l unilateral loss of hearing or when the dow curve" audiogram [4] suggests such loss, for unilateral deafness are applied. The nt is blindfolded for all tests not requiring n or observation of the eyes. A trained tant is at hand. Necessary conversation is ucted in low tones, and unnecessary per- el is excluded.

t first a trained medical corpsman performed initial tests for unilateral deafness. This tice was discontinued when it became appar- that the first examination is often the best ortunity for detecting the malingerer. The asking of malingerers is unimportant and in- uent compared with the proper evaluation of al unilateral loss of hearing.

he tests finally settled on for estimating pres- and degree of unilateral deafness are dis- ed here. A form found useful in recording lts of tests is given in the appendix. Care- made records permit reconstruction of tests. arded tests are discussed later in this paper.

nconsistency in Results.[5]—This subject is t with in the discussion of the actual tests mphasize its importance. Harbert [1a] stated "the only consistent finding in simulated and tional deafness is the inconsistency." The truth of this observation becomes apparent as one examines patients for unilateral deafness. One gradually realizes that the very inconsistency of the results is of great diagnostic significance. Some patients must be tested repeatedly to establish the truth about their hearing.

Shadow Curve Audiogram.—Bunch's [4] textbook states that "patients claiming total unilateral deafness must give a shadow curve when the ear claimed to be deaf is tested." The sounds of the audiometer are heard in the opposite ear. The patient must reproduce the shadow curve on repeated tests within limits of error. The shadow curve is from 40 to 60 decibels below the curve of the better ear but roughly parallel to it. If the level of hearing in the better ear is relatively normal and no shadow curve is registered for the poorer ear, the patient is malingering. The patient should be instructed to answer when he hears the test sound, regardless of which ear perceives it.

Wells-Stenger Test.—Several satisfactory examination procedures for use when unilateral deafness is suspected are based on the principle that one localizes sound toward the ear in which it is loudest. One is not aware of the same sound arising simultaneously but with less intensity from another source near the opposite ear. Any one with normal hearing can prove this to his own satisfaction by connecting the stem of a tuning fork to a rubber tube and then holding the vibrating fork about 7.5 cm. (3 inches) lateral to one ear and placing the open end of the tube in his other ear. He cannot hear the fork in the ear to which it is closer as long as the tube conducts the sound to the opposite ear. When the tube is removed from the ear, the fork is immediately heard and its position correctly estimated. As the auscultatory tube is removed and replaced in the ear, one feels that the source of sound is alternately on the right and left sides of the head.

The principle just discussed is applied in the classic Bloch-Stenger [6] test. The modification by Wells [7] has proved useful. The test is performed as follows: A tuning fork is selected which has a frequency well heard by the patient's better ear as determined by the audiogram. The stem of the fork is connected to a rubber tube about 75 cm. (30 inches) long and about 0.6 cm.

Mackenzie, G. W.: Simulated Unilateral Deaf- Laryngoscope **39**:103 (Feb.) 1929.

6. (*a*) Kerrison.[1d] (*b*) Stenger, P., cited by Grove.[2b] (*c*) Sonnenschein, R.: Functional Examination of Hearing, Arch. Otolaryng. **11**:97-116 (Jan.) 1930. (*d*) Mackenzie, G. W.: An Infallible Test for the Detection of Simulated Unilateral Deafness, Eye, Ear, Nose & Throat Monthly **18**:361 (Jan.) 1940.

7. Wells, W. A.: A New and Simple Method of Detecting Feigned Unilateral Deafness, J. A. M. A. **81**:199-200 (July 21) 1923.

(¼ inch) in diameter. An ear tip is inserted into the open end of the tube. The fork is struck, and the patient is instructed that his "hearing" ear is to be tested. The distance at which the fork is heard is measured and recorded on the examination form. The rubber tube is then connected to the "deaf" ear, and the patient is asked if he hears the sound when the fork is struck outside the range of the "hearing" ear. If he is deaf or malingering he denies hearing the tone. The fork is then moved into the hearing range of the better ear. If the poorer ear is totally deaf, or nearly so, the hearing distance of the better ear will be about the same as previously determined. If the patient is malingering, he will hear the fork immediately in his "deaf" ear but must wait till it is close enough to be perceived in his hearing ear before he can admit hearing it. Again the distance is recorded. The test is repeated to make sure of the result. The audiometer may be used as a source of sound [8] but is more complex than necessary.

Lombard Test.[9]—The Lombard test employs the principle that the loudness of the speaking voice is governed according to the way it sounds to the speaker. When a person having normal binaural hearing is speaking aloud and temporarily loses the hearing in one ear, he raises his voice immediately. The increase in volume is maintained until he again hears with both ears. Such temporary loss of hearing can be produced by sounding a Bárány noisemaker in one ear.

When the Bárány noise apparatus is sounded in the hearing ear of a unilaterally deaf person while he is speaking, he raises his voice, but he raises it to a greater degree than the person with normal binaural hearing. When the masker is sounded in a truly deafened ear, no change in volume of speech occurs.

The application of this test to soldiers having different reading aptitudes is facilitated through having reading material of varying content at hand. This may range from children's story books to daily newspapers. It is essential that the person being tested read steadily, to establish a rather constant level of speech volume. Changes on application of the masker are then clearly apparent. It has been claimed [1b] that malingerers can raise and lower their voices and outwit the examiner by giving correct responses. This may be true now and then, but the test has been tried on thoroughly co controls and has been found generally re

Marx Test.[10]—This simple test is extre useful. A Bárány noise apparatus is sho the patient. It is turned on, and the pati informed that his "good" ear is to be te He is asked to indicate which ear is the be The examiner stands behind the patient to clude lip reading. When the patient has subtly convinced that only his hearing ear be tested, the noisemaker is set going in ear. He is asked whether he hears the n If he answers, he is shown to be a m gerer, because the hearing ear is compl masked. The Marx test can be performed the Lombard reading test is being done. W the patient reads with his good ear masked is asked to stop and read from another pa the page. If he unwittingly complies, h exposed. The stream of water used in the c test also makes an efficient masker.[1a] Inno queries regarding the degree of dizziness o times trap the suspect into speaking alth his better ear is masked.

AUXILIARY TESTS

The tests just described comprise the u examination routine. The following additi procedures are carried out when necessary.

Watch and Tube Test (Becker).[11]—This is similar to one described by Callahan.[6e] T procedure was given an extensive trial and entirely satisfactory with some patients. I performed by carrying the sound of a tic watch to both of the patient's ears at the s time by means of a rubber tube connecting two ears. The watch is pressed against the of the tube. The loudness of the sound in e ear is regulated by moving the watch nearer ear than the other. Sound transmission to e ear is interrupted by pinching the tube. The is based on the same principle as the Wells-Ste er test, described earlier in this paper. It w be completely satisfactory if it did not utilize high-pitched tones of a watch tick. Since m persons lose perception of high frequencies e the test is not always applicable.

The Hummel Test (Double Conversa Test).[12]—A rubber tube 180 cm. (6 feet) l and 0.6 cm. (¼ inch) in diameter is connec

8. Guttman, J.: A New Method of Determining Unilateral Deafness and Malingering, Laryngoscope **38**: 686 (Oct.) 1928.

9. (*a*) Kerrison. [1d] (*b*) Grove.[2b] (*c*) Asherson, N.: A Test for Functional Aphonia and for the Detection of Complete Unilateral, Feigned Nerve Deafness, J. Laryng. & Otol. **51**:527-529 (Aug.) 1936.

10. Denker, A., and Kahler, O., cited by Grove.[2b]

11. Becker, B. M.: A Simple Method for Exp Malingering in Unilateral Deafness, Arch. Otola **9**:440-441 (April) 1929.

12. (*a*) Grove.[2b] (*b*) Fowler, E. P.: Medici the Ear, New York, Thos. Nelson & Sons, 193 374-377.

h ear. A glass funnel is used for a mouth-
at the opposite end of each tube. A short
nce whispered into the tube leading to the
ng ear is repeated by the patient. A
nce whispered by a second speaker into the
connected to the "deaf" ear is not repeated
e patient. A truly deaf person does not
and a malingerer denies that he is hearing.
examiners then whisper into their respec-
mouthpieces at the same time, and the
nt is asked to repeat what he hears. If he
hearing in both ears he is supposed not to
t, owing to his confusion. If he hears with
ear only, he repeats what was spoken into
ear. This test is often satisfactory, but a
l listener can concentrate on one ear.
the test is to be employed, several pre-
ons should be observed. The voice speaking
the "deaf" ear should be louder and should
before and end after the voice talking to
good ear. The two speakers should have
en material to read and should rehearse
to the test, using a person familiar with the
s their subject.

one and Air Conduction Test (Becker).[13]—
ubber tube about 75 cm. (30 inches) long
ng a diameter of about 0.6 cm. (¼ inch) is
d at one end. A loop is made at that end
e enough to admit the stem of a tuning fork
out compressing the lumen. The loop is
by a rubber band. An ordinary ear tip is
ted into the opposite end of the tube. A fork
ng a period between 128 and 512 double
ations per second is used. The test is based
he principle that normal hearing by air con-
ion is of greater intensity and duration than
ing by bone conduction. The author claims
hearing by tube conduction as used in his
is greater than hearing by direct air conduc-

he normal ear is tested by placing the stem
he vibrating fork on the mastoid bone. The
ent raises his hand when he hears the sound
lowers it when the sound dies out. The time
ecorded in seconds. Three or four trials are
e. The air conduction time for the normal
is ascertained next. The stem of the fork is
rted through the loop of the rubber tube and
ear tip is introduced into the "good" ear.
fork is struck, and the time until the sound
es is noted for several trials.

he test proper is conducted with the handle
he fork placed through the rubber loop. The
tip is inserted into the "deaf" ear, and the
fork is struck. The stem of the fork is then placed on the mastoid bone of the hearing ear. The tube should be connected to the "deaf" ear before the fork is placed near the good ear. This prevents the subject from surmising that the source of sound is actually near the good ear.

Several responses can prove malingering. The patient may disclaim all hearing, believing that sound is being carried only to his "deficient" ear. He may indicate hearing in his "deaf" ear if he is very shrewd. The bone conduction time for several trials must then correspond to that obtained in the preliminary bone conduction tests. He may be deceived by variation in the vibration time of the fork. Several sets of normal values may be set up during the preliminary testing through use of a single fork struck blows of unequal intensity. Certain other possible responses suggested by Becker have not been helpful.

Teal Test.[14]—When a patient denies air conduction but admits bone conduction in the affected ear, this test may be used. An inactive fork is placed against the mastoid process of the "deaf" ear just as a vibrating fork is brought near to the same ear. The subject may conclude that the fork on the mastoid bone is vibrating and admit that he hears. He thereby involuntarily indicates that he can hear by air conduction with his so-called deaf ear.

Caloric Test.[1d]—The usual routine of irrigating the ear canal with cold water may be done to induce nystagmus and past pointing. Absence of normal response is not pathognomonic of total deafness but is strong collateral evidence.

TESTS DISCARDED AFTER TRIAL

Several of the tests discarded during evolution of the examination routine involve ostensible occlusion followed by surreptitious opening of the ear canals with the resultant influence on hearing. The modus operandi varies.

Tests Involving Ostensible Occlusion.—The Tschudi test[10] employs four aural speculums. Two are filled with wax. In the Stethoscope test[15] the ear pieces of an ordinary binaural stethoscope are placed in the patient's ears and one tube is occluded by clamping or plugging. Sound is delivered to the chest piece of the stethoscope. This test has proved entirely valueless. Others[16] have reported similarly. The apparatus is not "sound tight," and sounds enter-

13. Becker, B. M.: A Combined Air and Bone Conion Test for Pretended Deafness, Arch. Otolaryng. 214-218 (Aug.) 1934.

14. (*a*) Harbert.[1a] (*b*) Lewy, A., and Shapiro, S. L.: The Functional Examination of Hearing, Arch. Otolarying. **39**:268 (March) 1944.

15. (*a*) Harbert.[1a] (*b*) Kerrison.[1d] (*c*) Grove.[2b]

16. (*a*) Harbert.[1a] (*b*) Grove.[2b]

ing the stethoscope bell are heard through air outside the tubing. Accurate localization of sound seems impossible, perhaps owing to the conduction of sound by metal from one ear to the other. A stethoscope having unequal lengths of tubing leading from the ear pieces to the bell is sometimes employed. Localization of sound is supposed to depend on the damping effect of the longer tube.[17] This test has all the faults of the ordinary stethoscope test. In the Megoni test [18] the good ear is closed by the examiner's finger while words are whispered into the "deaf" ear. The good ear is unstopped suddenly as the whispering continues. If the patient denies hearing, his malingering is exposed, since his hearing ear is no longer occluded. The Dölger test [19] assumes that the patient hears the same intensity of voice or whisper at twice the distance with the examiner's mouth turned toward him as with the examiner facing away from him. This assumption ignores the reflection and absorption of sound and therefore is not well founded.

In the Erhard test [2b] the subject is asked to count watch or metronome ticks or to repeat conversation with the hearing ear open and the "deaf" ear closed. The hearing ear is then shut with the finger and the source of sound moved close enough to be audible despite the closed meatus. The malingerer may disclaim hearing, believing that closure of his auditory canals should make him totally deaf.

In all the tests just described one common weakness occurs. The patient ought to hear something, but he says that he does not. His denial is worth just as much as the examiner's statement that he should have been able to hear. A conclusive test for malingering should cause the subject to perform an overt act when he hears the sound he pretends not to hear. The tests included in the routine examination meet this specification. Inconsistency in the results of tests and failure to record a shadow curve with the audiometer [4] are matters of record. The changes in distance in the Wells-Stenger test [7] are grossly measurable. The amplitude of the voice in the Lombard test [9] is very apparent. When the patient answers in the Marx test,[10] the presence of hearing in his "deaf" ear is obvious.

Several other tests were tried but were not adopted for routine use. These are described in following paragraphs.

17. Becker, B. M.: A Novel and Simple Method for the Detection of Unilateral Deafness, Laryngoscope **38**:677 (Oct.) 1928.

18. Megoni, S., cited by Grove.[2b]

19. Dölger, cited by Grove.[2b]

Chimani-Moos Test.[20]—A vibrating tu fork of 256 double vibrations per second is pl on the vertex of the skull, as in the stan Weber test. The suspect is said to be malinge if he lateralizes the sound toward the affecte with a perceptive lesion or toward the good with a conductive lesion or if he does not lat ize. Any one who has performed a large num of tuning fork tests knows that many pati give unexplained atypical responses. As Bunch says, "The examiner who makes a diagnosis malingering on the basis of some atyp response to the Weber test may find himself a position very difficult to maintain."

Wotzilka Lid Movement Reflex Test.[20b]—T subject is directed to look upward while examiner stands to the side and observes his lids. A high-pitched fork is struck near "deaf" ear. A sharp reflex wink is supposed occur if the sound is heard. This test was tr on medical corpsmen and officers whose hear was normal. One ear was masked. The respo was difficult to see and to distinguish fr ordinary winking. A variation of this pro cedure is to place biconvex lenses before patient's eyes and observe dilation of the pu when the sound is heard.[21]

Nadoleczny Test.[2b]—The patient lip re numbers spoken by the examiner. An assist whispers different numbers into the patie "deaf" ear. If he is really deaf, he sho perceive only the number spoken by the exa iner. If not deaf he may repeat the num spoken by the assistant. This procedure is ti consuming and requires a fair degree of skill the part of the patient. Furthermore, the pati can invalidate the test by claiming to have read the number spoken into his ear.

SUMMARY

Tests for unilateral deafness that have be found useful in a military hospital have evol into an examination routine to include the follo ing procedures: (1) shadow curve audiogra (2) Wells modification of the Stenger test, Lombard reading test and the (4) Marx te Certain other procedures are used as auxili tests. These are: (1) watch and tube t (Becker), (2) Hummel double conversation te (3) bone and air conduction test (Becker), Teal test and (5) caloric test (Bárány).

Tests found unsuitable for the usual exami tion routine include those which involve os

20. (*a*) Fowler.[12b] (*b*) Bunch.[4] p. 18.

21. (*a*) Fowler.[12b] (*b*) Unger, M.: Objective M surement of Hearing, Arch. Otolaryng. **29**:62 (April) 1939.

occlusion followed by surreptitious opening e auditory canals with the resultant effect aring. Other tests which were tried but dis- d were the Chimani-Moos test, the Wotzilka ovement reflex test and the lip reading test adoleczny.

APPENDIX

TESTS FOR UNILATERAL DEAFNESS

ROUTINE TESTS

..... Rank..... Date.....
nization..... A.S.N..... Age.....
for Unilateral Deafness: Patient claims.....
(total, partial)
ness in.....ear

D I
1. Shadow curve audiogram.....
(yes, no)

D I 2. **Wells Modification of Stenger Test**
Tuning fork with rubber tube attached to stem
a. Fork heard in hearing ear....inches (Tube not connected to either ear)
b. Fork heard in hearing ear....inches (Tube connected to poorer ear).....fork used
Remarks .....

D I 3. **Lombard Reading Test**
With Bárány masker in hearing ear, patient .....raise voice while reading aloud
(does, does not)
With Bárány masker in nonhearing ear, patient.....raise voice while reading aloud
(does, does not)
Remarks .....

D I 4. **Marx Test**
Bárány masker in hearing ear, patient.....
(does, does not)
answer with hearing ear totally masked
Remarks .....

AUXILIARY TESTS

D I 5. **Watch and Tube Test (Becker)**
a. With....inch long tube connected to hearing ear, patient heard watch at....inches through tube
b. With tube connected to both ears and watch placed....inches from hearing ear, patient heard .....
atch in good ear, watch in bad ear, did not hear watch)
c. With tube to good ear pinched, patient .....hear watch
(does, does not)
Remarks .....

H D I 6. **Hummel Test (double conversation test)**
Patient repeats right ear.....Patient repeats
(yes, no)
left ear.....
(yes, no)
Remarks .....

H D I 7. **Bone and Air Conduction Test (Becker)**
a. Better ear: Air (through tube) ... ...
1 2 3
trial (seconds)
Bone ... ... trial (seconds)
1 2 3
b. With tube in "deaf" ear and fork on better mastoid, patient raises hand on side of .....
(hearing ear, deaf ear, denies all hearing)
c. If hearing in deaf ear (bone conduction) indicated, time ... ... trials.
1 2 3
d. With inactive fork on "good" mastoid and live fork against tube, patient admits hearing .....
(yes, no)

H D I 8. **Teal Test**
With inert fork on deaf mastoid and active fork near pinna, patient hears .....
(yes, no)
Remarks .....

H D I 9. **Caloric Test (Bárány)**
Right ear irrigated.....with water at 68°F.
seconds
caused nystagmus.....
(yes, no)
Quick component to.....Past pointing to
(left, right)
.....
(left, right)
Left ear irrigated.....with water at 68°F.
seconds
caused nystagmus.....
(yes, no)
Quick component to.....Past pointing to
(left, right)
.....
(left, right)
While good ear is irrigated patient replies to questions.....
(yes, no)
Remarks .....
Examiner.....

NOTE.—The letters H, D, I in the margin stand for the words honest, dishonest and inconsistent. The appropriate letter is encircled after the test is completed. One can glance quickly at the encircled letters and determine the result of the examination.

DEAFNESS: ITS CAUSES AND WHAT CAN BE DONE ABOUT IT

M. H. LURIE, M.D.
BOSTON

Eleven years ago, at a meeting of the American Academy of Ophthalmology and Otolaryngology, I read a paper entitled "Animal Experimentation on Hearing: Its Clues to the Prevention of Deafness."[1] Since then a great deal of experimentation on deafness has been done with both animals and human beings as subjects. In the past three years extensive experimental work has been carried on as a result of demands for answers to problems raised by the war. Again the question is asked, "What is deafness, and what can otologists do for it?"

This question divides itself, as always, into two components: (1) the problem of deafness due to mechanical causes, that is, to obstructive conditions that prevent sound from reaching the sense organ or the organ of Corti (middle ear deafness), and (2) the problem of deafness due to changes occurring in the sense organ and the cochlear nerve (nerve deafness). These two components rarely occur as separate entities. One finds various combinations of the two, with one or the other tending to predominate. It is only in cases of acute infection of the middle ear that one can say a condition of mechanical deafness due to infection prevails. The deafness secondary to mumps may be considered a true sense organ or nerve deafness.

The problems of the deaf person are usually complex and at times seem insoluble. From the experimental point of view they are extremely difficult, and certain ones will, in all probability, never be solved. In animals otologists can produce lesions of the sense organ or of the conduction apparatus and demonstrate them microscopically, but they cannot tell accurately what hearing was lost by these animals as a result of their experiments. In human beings they can cause acute losses of hearing by various means, but they cannot demonstrate what has happened or what may happen to the sense organ. Therefore, one must try not to be dogmatic in one's theories and views on deafness.

Deafness of the mechanical type (middle ear deafness) includes all types of pathologic changes which prevent sounds from reaching the sense organ. Thus into this group go all the known causes of middle ear deafness, such as retraction of the drum, granulations and adhesions of the drum to the inner wall of the middle ear secondary to infection, fusion of the malleus and incus and fusion of the stapes to the oval window. For these conditions various treatments have been advised, and it is not necessary to review them at this time. They all have their value, but, unfortunately, they have as a general rule given only temporary relief. The reason why relief is only temporary is that one cannot in most instances remove or by-pass the fundamental cause of the condition causing the deafness.

It is in the treatment of deafness due to otosclerosis, fusion of the stapes to the oval window and fusion of the malleolus and the incus that the greatest advances have been made. I refer to the fenestra operation of Lempert.[2] This is a technic that has been developed to bypass the mechanical difficulties caused by the disease. I shall not enter into any discussion as to the value of the fenestra nov-ovalis in place of a hearing aid or consider whether or not fenestration is still in the experimental stage. It is an operation that will find its own place in the practice of otology; many of the difficulties connected with it at the present time will surely be overcome as otologists have more experience with it. The operation is not a cure for the disease that is present; what it does is to enable sound to reach the organ of Corti. The failures of the operation are due to changes caused in the labyrinth by the operation itself—overgrowth of scar tissue and regeneration of bone at the point of fenestration. This fact indicates a need

From the Departments of Otology and Laryngology, Harvard Medical School.

Presented at the meeting of the American Academy of Ophthalmology and Otolaryngology, at Chicago, Oct. 11, 1944.

1. Lurie, M. H.: Animal Experimentation on Hearing: Its Clues to the Prevention of Deafness, Tr. Am. Acad. Ophth. **40**:375, 1935.

2. Lempert, J.: Fenestra Nov-Ovalis: New Window for Improvement of Hearing in Cases of Otosclerosis, Arch. Otolaryng. **34**:880 (Nov.) 1941.

careful selection of subjects for operation thorough study of the failures. It is from failures that investigators will learn most; successes convey little information.[3]

To the person with middle ear deafness little can be offered at the present time except haps the improved hearing apparatus being loped as a result of the war.

deafness caused by changes in the sense n, again there seem to be many causes with same end result—impairment of hearing of ous degrees. At the present time, as the lt of the experimentation on man and ani-, it is my belief that this type of deafness rimarily due to degeneration of the organ orti. In animals, otologists, using loud and nsive sounds of short duration, have been to produce lesions of the organ of Corti ing from immediate rupture and destruction e organ to degeneration occurring days later arts of the cochlea not affected by the acute osure. Experimentation on man tends to out the experimentation on animals, pro- g evidence that nerve deafness is due primar- to changes occurring in the organ of Corti. m the work on animals it appears that changes rring in the cells of the organ of Corti are rsible if they do not go beyond a certain t. Once this point has been passed, the cells enerate and the organ of Corti does not func- normally.

rugs can cause similar degenerative changes, as been reported in previous articles.[4] Covell[5] rted changes in the organ of Corti in avita- osis. In animals experiments with contin- s or interrupted sounds have given similar lts.[6]

n experimentation on human beings, as in on animals, evidence has been produced there is a great variation in the reactions ndividual ears to intense sounds. One person may be greatly affected for a long period, while another person subjected to the same exposure may show little after-effect. One person makes a quick recovery and another a slow recovery. These observations lead one to an interesting speculation in regard to the organ of Corti. Is there in the organ of Corti a chemical mediator that is essential for hearing? Does the organ lose this chemical mediator quickly on exposure to sounds or to disease and, not being able to restore it, does the organ begin to degenerate? There is some evidence pointing to a chemical mediator, but a great deal of study and biochemical investigation will have to be done before it can be proved that such a factor is at work. Otologists do not know why there are these tremendous variations and degrees of nerve deafness among people. It must be confessed that they know little about how the organ of Corti functions. This means that the treatment and the prevention of nerve deafness cannot be established on a firm foundation. Otologists know that exposure to sounds of certain intensities causes deafness in some persons and not in others, that disease causes deafness in some and not in others and that one person can take quinine for long periods without any effect on his hearing, while another loses his hearing quickly if he takes quinine. What is the factor that causes these variations? Not knowing, otologists cannot intelligently treat the person with nerve deafness.

The use of vitamins to build up a person's general health is justified, but their use as a cure for nerve deafness represents a delusion. Once deafness has appeared so that the patient complains about it, it indicates, in my opinion, definite pathologic changes in the organ of Corti, and nothing will reverse these changes. The same reasoning holds for the use of endocrine substances; I have not seen any cures of deafness from endocrine therapy.

The treatment of the person with nerve deafness, as I see it at present, is to keep his general health up to its maximum level, help him with a hearing aid, reeducate his ears and stress lip reading if he is young enough to take it up. Otologists have nothing to offer him except from the point of view of psychology and rehabilitation. I know of no drugs that will cure nerve deafness.

I purposely present this pessimistic view, for in the next five to ten years there will be many young persons with nerve deafness as a result of the war. Criticism of the handling and treatment of these patients will certainly increase as

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Lempert, J.: Lempert Fenestra Nov-Ovalis with ile Stopple: A New Advance in the Surgical Treat- for Clinical Otosclerosis Evolved as a Result of esearch Study of One Thousand Cases in Which estration Has Been Performed During the Last n Years, Arch. Otolaryng. **41**:1 (Jan.) 1945.

Lurie, M. H.: What Is Perception Deafness a Physiological and Histological Basis? Ann. , Rhin. & Laryng. **48**:3 (March) 1939.

Covell, W. P.: Pathologic Changes in Peripheral itory Mechanism Due to Avitaminosis, Laryngoscope 32 (July) 1940.

Davis, H.; Derbyshire, A. J.; Kemp, E. H.; e, M. H., and Upton, M.: Functional and Histo- al Changes in the Cochlea of the Guinea Pig ulting from Prolonged Stimulation, J. Gen. Psychol. 251, 1935.

time goes on. There will be enthusiastic advocates for various treatments and for special drugs. Many persons will complain that nothing is being done to help the deaf. The truth is that a great deal will be done for the deaf, but it will not be done by otologists. Psychologists, teachers of lip reading and physical therapeutists reeducating the deaf will play the principal roles in the handling of these patients. Also, it must be acknowledged that, not knowing the cause, we cannot honestly offer any specific method or cure for nerve deafness. Otologists should recognize this fact and do all they can to encourage and continue research studies that they believe will result in help for the deaf.

483 Beacon Street.

Clinical Notes

TREATMENT OF EXTERNAL OTITIS

JOSEPH GOLDSTEIN, M.D.
LOS ANGELES

The literature on the treatment of external otitis, especially that of otomycosis, was recently carefully reviewed by Senturia and Wolf.[1]

Anatomically the external auditory canal is analogous to a test tube in an incubator, with a medium of cerumen and exfoliated epithelium. The body supplies moisture and an even temperature. The canal is easily infected and difficult to treat because of inaccessibility of infected parts.

Mixtures of fungi, molds and bacteria invade the normal glands. They irritate the membranous canal wall, causing edema. The patients complain of itching and proceed to scratch. This brings on an inflammatory reaction with an increase of edema until at times the external auditory canal may become occluded. I have observed cases in which the tympanic membrane was perforated and the middle ear involved by infections of the external auditory canal. More often one sees recurrent otitis media macerating the canal walls and these canal walls later infected by the various fungi producing external otitis.

1. Senturia, B. H., and Wolf, F. T.: Treatment of External Otitis: II. Action of Sulfonamide Compounds on Fungi Isolated from Cases of Otomycosis, Arch. Otolaryng. **41**:56-63 (Jan.) 1945.

Numerous types of therapy give unsatisfactory results. I offer an original treatment which has proved successful.

TREATMENT

A solution is made by dissolving 1 chloramine (chloramine, U. S. P.) tablet in 1 ounce (29.5 cc.) tepid water.

The patient lies on his side with the affected ear up. Using an ordinary medicine dropper, the otologist flushes the auditory canal, alternately filling the canal and withdrawing the liquid and debris. When the canal returns clear, the canal is filled and the solution allowed to remain a few minutes; then the ear is dried. It is permissible to put liquid petrolatum on the macerated skin. This is not necessary until the end of the course of treatment, since the solution does not abraid the skin.

The patient should return on alternate days until the ear is dry, the course taking usually less than ten days. He does not complain of discomfort during the treatment. He usually is at ease the first night and hears without complaint of a muffled tone.

In a few days the canal is dry and the itching has disappeared. The otologist then swabs the canal with a solution of metacresylacetate (cresatin) to which is added thymol in a concentration of 1 per cent. If the skin is too dry after the treatment, the ear may be swabbed with petrolatum.

727 West Seventh Street.

Case Reports

PENICILLIN THERAPY FOR THROMBOSIS OF THE CAVERNOUS SINUS IN A FOURTEEN MONTH OLD CHILD

J. WARREN WHITE, M.D., NORFOLK, VA.

For a long time it has been known that the ernous sinus is vulnerable to infections of face and the nose. Thrombosis of the ernous sinus was usually considered a fatal se until the advent of sulfonamide com- ds in therapy. Recovery was rare.

rove [1] after an extensive review of the litera- on the subject in 1936 made the following ments: "In the septic types in which the mbosis or thrombophlebitis reaches the cav- us sinus by way of the afferent vessels the tality is practically 100 per cent. . . . When hrombus originates from a septic source, the should be vigorously treated. The cavern- sinus itself should be let alone."

. 1.—The patient as he appeared twenty-four hours the beginning of treatment with penicillin.

Staphylococcus albus and Staphylococcus us are the organisms usually responsible for thrombosis. Thrombosis of the cavernous s can occur at any age but the youngest nt with recovery whose case I have found orted (by Cohen and Cohen [2]) was aged 2 years and 3 months. The present case is interesting for several reasons. First, the child was only 14 months old; second, no response to the sulfonamide compound was observed; and third, prompt recovery occurred after treatment with penicillin.

REPORT OF CASE

E. S., a boy aged 14 months, was admitted to Norfolk General Hospital, Dec. 24, 1943, with the following history: Ten days previously an illness developed which was diagnosed as bronchopneumonia. A few days before entering the hospital he had several convulsions and swelling of the left eyelids. A consultation was held, and it was decided that involvement of the cavernous sinus was the disturbing factor.

When I first saw the baby he had the appearance of being exceedingly ill. The right eye was normal. The left eye was proptosed and completely closed by a much swollen upper lid, making it difficult to get a view of the eye. He had a profuse purulent discharge from the left nostril and some edema over the left antrum. The report on the physical findings was that he had numerous moist rales scattered throughout both lungs. The hemoglobin content was 69 per cent; the red blood cell count, 3,510,000; the white cell count, 17,800, with polymorphonuclears 73 per cent and small lymphocytes 24 per cent. Urinalysis showed a trace of albumin.

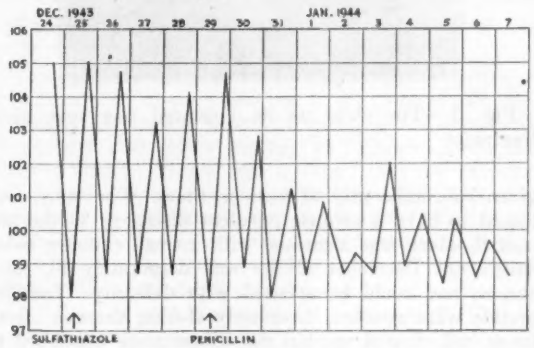

Fig. 2.—Temperature curve.

The report of the roentgen examination (Dr. P. B. Parsons) follows: "Posteroanterior projections of the sinuses show that the left maxillary antrum is entirely opaque. The left nostril contains no air. The nasal septum is in the midline. A roentgenogram of the chest shows heavy peribronchial thickening with some patchy consolidation throughout both lung fields. The heart and the great blood vessels are within normal limits of size and shape. The diaphragms are smooth. Impression: (1) sinusitis, antral, left; (2) bilateral bronchopneumonia."

Read at the meeting of the Southern Section of the erican Laryngological, Rhinological and Otological ety, at Charlotte, N. C., Jan. 15, 1945.

1. Grove, W. E.: Septic and Aseptic Types of mbosis of Cavernous Sinus, Arch. Otolaryng. **24**: July) 1936.

2. Cohen, M., and Cohen, H. J.: Septic Thrombo- itis of the Cavernous Sinus in a Two Year Old Child, Am. J. Dis. Child. **63**:546 (March) 1942.

The temperature was fluctuating daily between normal and 104 and 105 F. Sulfathiazole was given, and nasal drops of a suspension of 6.25 per cent sulfanilamide and 6.25 per cent sulfathiazole (Duosulfon Suspension) were used in the left nostril. The blood concentration of sulfathiazole was 6.6 mg. per hundred cubic centimeters on December 27. Culture of the pus obtained from the left nostril yielded a pure growth of hemolytic Staph. aureus, and on the next day culture of the blood yielded a growth of Staph. aureus. Little nourishment could be given by mouth. A 5 per cent solution of dextrose was

Fig. 3.—The child as he appeared one year after treatment.

given subcutaneously at various times. The baby continued to have a profuse purulent discharge in the left nostril, which was aspirated with a small catheter twice daily. On December 29 he was desperately ill, in a stupor, and could be aroused with difficulty. The left eyelids were swollen, completely closing the eye. Both lungs had cleared up, but the temperature continued to fluctuate between normal and 104.6 F. The baby's father was in the United States Army, and at the suggestion and through the efforts of Dr. Claiborne Willcox, penicillin was sent from Boston by plane. At 2 p. m. December 29 treatment with penicillin was begun; 5,000 units was given intramuscularly every three hours January 1 and then 2,500 units every three hours January 7. On December 30, the day after pe therapy was begun, the baby began to take nouris and the temperature dropped and did not again go 102.4 F. December 30 a culture of blood from the stream was negative, and from this time on was rapid convalescence with complete recovery no apparent damage to the eye. The patient wa charged from the hospital two weeks after st to receive penicillin. It was dramatic to see a desperately ill and then, in twenty-four hours the beginning of the administration of penicillin, t nourishment and playing with toys in his crib.

On Jan. 2, 1945, he had vision in the left eye, b was impossible to make an accurate test as to much. The ophthalmoscopic examination revealed ing abnormal, and the eye was normal in appea

COMMENT

From a clinical standpoint the child suf from thrombosis of the cavernous sinus, the diagnosis was based on: (1) a tempera fluctuating daily between normal and 10 105 F.; (2) a blood culture yielding St aureus; (3) an acute purulent discharge the left nostril (the left antrum was the s source of the infection); a culture of the yielding a pure growth of hemolytic St aureus; (4) cultures of the pus obtained f the left nostril and of the blood both yie the same organism, a pure growth of St aureus (the organisms recovered from the in the nostril were more actively hemolytic those recovered from the blood stream); (5 proptosed left eye with edema of the lids.

SUMMARY

A boy 14 months old who appeared al moribund with thrombosis of the left caver sinus showed almost no response under s thiazole therapy though the blood concentra of the drug was 6.6 mg. per hundred c centimeters. Under treatment with penicillin t was improvement within twenty-four hours, a negative blood culture and rapid convalesce the baby being discharged from the hos two weeks after this treatment was begun.

Abstracts from Current Literature

Ear

Otological Views. Adolf Schwarzbart, J. Laryng. & Otol. **59**:47 (Feb.) 1944.

warzbart presents his views on the origin, the pment and the function of the eustachian tube, the ear and the cells of the mastoid bone, based on gic and histopathologic studies of serial sections temporal bones and eustachian tubes. The genesis ome clinical aspects of disease of the middle ear so presented. He finds the established division of iddle ear cavity and eustachian tube (bony and aginous portions) an untenable concept. Since the portion of the eustachian tube is an anterior evagi- of the tympanic cavity, he considers it a part of iddle ear. For it the term "protympanum" is ed in analogy with the epitympanum and hypo- num. The entire tympanic cavity, containing the nic cavity, the protympanum (bony portion of the hian tube) and the pneumatic spaces, is referred to bitympanum.

author finds that the bitympanic spaces and their ng epithelial cells do not develop as usually sup- from the tubal duct with its entodermal epithelium, is the product of the first branchial cleft. De- ment of the epithelium of the bitympanic spaces is he mesenchyme and the marrow of the fetal tem- bone by (*a*) transformation of "rich-in-cells" w into "poor-in-cells" jelly-like tissue, (*b*) forma- of a cleft and (*c*) self epithelization (pneumatiza- of the cleft. An analogy is drawn by citing, for le, the formation of blood vessels from cleftlike chymal structures. Histologically, tubal epithe- is double-layered stratified ciliated cylindric epi- m with goblet cells, while bitympanic epithelium endothelium-like layer of flat epithelium.

ctionally, morphologically and embryologically ithelium of the tube resembles that of the mucous rane of the nasopharynx. On the other hand, the panic epithelium is formed by a thin endothelium- membrane acting only as a delicate separating between the acoustic air spaces and the periosteum temporal bone, to function as a sound-transmitting ound-transforming medium. Chemical, toxic and ious substances find it a poor defense, its reaction ting of transudation or exudation resembling that rue serous membrane. In maintaining the perfec- nd acoustic function of the bitympanum, the tube defensive and an equalizing function; the first is rmed by its mucous membrane with glands, lymph- ssue and cilia, while the second is performed by rtilaginous-muscular apparatus.

e author presents a new classification of cases of media, utilizing the anatomic divisions described. inflammatory diseases of the auditory tube are nated with the term "eustachitis" and those of the panum with "bitympanitis." The latter term em- s diseases of the endotympanum (endotympanitis), es of the pneumatic and marrow tissue (pneu- omyelitis) and diseases of the substance of the oral bone (osteitis). It is pointed out that eusta- and bitympanitis do not always occur in pure but are frequently associated with each other. Further, pneumosteomyelitis and osteitis have either a tubogenous or a hematogenous origin. While cases of tubal origin show a preceding endotympanitis and the usual picture of purulent otitis media, those of hematogenous origin do not always follow such a course and display a scarcity of otoscopic findings.

LeJeune and Steele, New Orleans.

The Treatment of Chronic Suppurative Otitis Media by the Local Application of Penicillin and Other Drugs. E. G. Collins and K. E. A. Hughes, J. Laryng. & Otol. **59**:81 (March) 1944.

Collins and Hughes present a series of 23 cases of chronic suppurative otitis media, in 9 of which penicillin was applied locally. The investigation involved examination of over 300 aural smears and 540 cultures, thereby permitting an interesting bacteriologic study of chronic suppurative otitis media. Rigid aseptic technic was followed in order to avoid meatal contamination, material for cultures and smears being taken by a platinum loop passed through the perforation into the middle ear. In the initial cultures, staphylococci were found three times, "coliform" bacilli ten times, Bacillus proteus thirteen times and diphtheroids six times, a mixed growth being common. It is believed that chronic suppurative otitis media furnishes an outstanding example of antibiosis.

Nine patients were treated by the local application of penicillin, 4 of these obtaining a dry and sterile ear. A similar result was observed in 12 of the remaining 14 patients after other therapy as follows: Local use of boric acid in 75 per cent alcohol, local use of boric acid–iodine powder, local application of sulfonamide compounds (sulfanilamide powder, succinylsulfathiazole powder, a 30 per cent solution of acetylsulfanilamide and a 15 per cent "cream suspension" of crystals of sulfathiazole [mikraform sulfathiazole]) and peroral administration of sulfathiazole in a dosage of 39 Gm. in seven days. It was felt that successful treatment of chronic suppurative otitis media was hindered greatly by the presence of "coliform" bacilli, B. proteus and diphtheroids. In some of the cases a 30 per cent solution of acetylsulfanilamide combined with penicillin proved to be fairly successful clinically in preventing this complication.

The authors emphasize the importance of daily careful toilet of the ear carried out by the otologist himself. Such care is believed to be in no small way responsible for obtaining dry, sterile ears for 70 per cent of the patients, including both the group treated with penicillin and those treated with other drugs.

The technic of the local use of penicillin, as well as its clinical and bacteriologic effects, is well illustrated with case records, the authors concluding that the types of organisms present and the difficulties of application will limit its use. Of all the bacilli found in the aural discharge, staphylococci and B. proteus are the greatest enemies, and when in combination they appear to be intractable to treatment. Penicillin was not used parenterally in this study.

LeJeune and Steele, New Orleans.

ROENTGENOGRAPHIC DIAGNOSIS OF COMPLICATIONS OF MASTOIDITIS. PEDRO REGULES and NICOLAS CAUBARRERE, An. de oto-rino-laring. d. Uruguay **14**:21, 1944.

The authors attempt to establish criteria for diagnosing the complications of mastoiditis. Their article deals primarily with interpretations of the roentgen findings, particularly those in the region of the lateral sinus. They describe what they term the gutter that runs anteriorly along the entire course of the sinus wall, and in the interpretation of the defects they believe that an alteration of the anterior wall of the gutter of the sinus is a positive sign of pathologic involvement of the sinus. In a group of 24 cases they were able to diagnose sinus disease preoperatively in 19 cases. The importance of this criterion is evident particularly when the symptoms are more or less latent, when the mastoiditis may be due to Pneumococcus mucosus or when treatment with sulfonamide compounds has been instituted and many of the findings are masked. In such cases prognosis is always graver and surgical intervention more urgent.

PERSKY, Philadelphia.

Pharynx

LATENT TUBERCULOSIS OF THE TONSILS. G. L. COTE, Union méd. du Canada **72**:16 (Jan.) 1943.

Tonsils from 60 patients from 4 to 27 years of age were examined histologically. Six patients were over 20, 14 were between 10 and 15, and 40 were from 4 to 10 years of age.

Roentgen study of the chest and gastric lavage with search for the tubercle bacillus had been performed on all patients. A tuberculous process was present in the tonsils of 8 patients (13.3 per cent). Two of these 8 patients were over 20 years of age; 6 had cervical adenopathy.

The author considers that tonsillectomy carries no risk and removes a focus (which is probably primary). He advocates routine examination of all removed tonsils to rule out latent, unsuspected tuberculosis.

DAFFINEE, Melrose, Mass. [AM. J. DIS. CHILD.]

Larynx

A REVIEW OF THE AVAILABLE LITERATURE ON THE LARYNX AND LARYNGEAL SURGERY FOR 1944. HENRY BOYLAN ORTON, Laryngoscope **55**:33 (Feb.) 1945.

The first aid treatment of laryngeal and cervical wounds is directed toward stopping hemorrhage and maintaining an airway. Digital pressure and ligation of the arteries may control the hemorrhage. Tracheotomy may be indicated. Later more extensive surgical treatment, including the repair of severed nerves, should be instituted. The mortality from penetrating wounds of the neck is high.

Biggs's paper on asphyxia neonatorum is outlined. Warmth, the Drinker respirator and keeping the air passages clear are important in the treatment of severe asphyxia. Congenital laryngeal stridor is discussed. Schwartz points out that "inspiratory laryngeal collapse" is a better term for this condition.

Several articles on the action of the laryngeal muscles are mentioned.

There have been several papers on laryngeal stenosis due to bilateral abductor paralysis. Means of alleviating this condition are outlined.

Papilloma and cystadenoma of the larynx are discussed.

Orton mentions several articles concerning cancer of the larynx. These deal particularly with the treatment. There is always the question of irradiation versus surgical removal, and if the latter, just what operation should be used. A radiotherapeutic test may be of value in deciding between operation and radiation in difficult cases.

HITSCHLER, Philadelphia.

Miscellaneous

PERSONALITY FACTORS IN A MOTOR SPEECH DEFECT CASE. C. M. RUSSELL, Am. J. Ment. Def. **49**:171, 1944.

A boy 5 years old did not talk. He had been previously psychometrically recorded, the terms ranging from "imbecile" to "bright and intelligent." He had very poor lingual and speech motor function, facial and bodily tics and an attitude of extreme negativism. Hearing was normal; the stature was normal; there were no gross organic findings. His one response was "ngah," and for a long time every new sound was prefaced by this response. When he began to learn, he had delayed responses, backward speech and stuttering. He finally recovered speech, had a good school record and was present attending a college. The author states that the work with this patient calls further attention to the vast amount of research and experimentation necessary on certain types of children who do not appear to have normal intelligence or any possibility of social maturity.

PALMER, Wichita, Kan.

NASAL INJURIES IN DYSTOCIA. MATTHEW S. ERSNER, Ann. Otol., Rhin. & Laryng. **53**:552 (Sept.) 1944.

Ersner discusses a situation which has been present since births began but which has only recently been recognized as a definite factor in the development of septal deflections and nasal obstruction. He points out that during the birth of a child a tremendous pressure (up to 30 or 40 pounds [13.5 or 18 Kg.]) is exerted by uterine contractures on the child, forcing it violently through the pelvis. With Dr. N. Kendall, of Tulane University Hospital, in studies of measurements of newborn infants, he found that there was a variation from 1 to 2.5 cm. of outward projection of the face. In only 1 child, a Negro, was there an exception, and here the occipitofrontal and occipitonasal measurements were the same. With a nasal projection that normal the nose is bound to receive considerable trauma in passing through the birth canal with so great pressure behind it, and this trauma is increased in dystocia with a large head in relation to the pelvis. The most common injury to the nose as a result is a dislocation of the cartilaginous portion of the septum from the bony groove. Rarely does the obstetrician look for intranasal injury, and such injury is thought of only after it is noted that the child has difficulty in nursing as well as in breathing through the nose. Ersner suggests that every obstetrician should routinely inspect the infant's nose for septal deflections or nasal and septal dislocations and fractures, just as routine examinations are conducted to determine the presence of any other deformities or injuries. It is best to correct the deformity immediately before a greenstick fracture has set or a callus forms. Reduction is readily done by grasping the tip of the nose and gently but firmly moving it from side to side or by tugging the tip of the

ventrally. Usually one can feel the cartilage to place on the vomer. In more difficult cases ments may be used to grasp the cartilage. If a e or a dislocation is neglected or overlooked ere is a lapse of several weeks, the additional n of callus develops. If the condition is entirely ked, one may find in later life a deflected septum asal obstruction; trigger points with resultant nerve involvement; faulty development of the jaw; sinusitis; the so-called adenoid facies due al obstruction; last but not least, the whole ound becomes a panorama of "noseology" at ence. As the nose begins to grow the external ity becomes apparent by distortion, asymmetry, ty or hump.

M. V. MILLER, Philadelphia.

TREATMENT OF RHINORRHEA AND OTORRHEA. E. DANDY, Arch. Surg. **49**:75 (Aug.) 1944.

brospinal fluid rhinorrhea and otorrhea result istulous openings between the subarachnoid or ular spaces and the accessory paranasal sinuses air cells of the mastoid bone. These conditions st frequently caused by fracture of the skull or ning created by operation; they are rarely due ion produced by tumor or infection or to a con- defect.

escape of cerebrospinal fluid may cease spon- ly, as it does in cases of otorrhea following e of the petrous bone. Leakage persisting longer wo weeks must be treated surgically, since sooner r meningitis or abscess of the brain will develop. the operation is practically free from danger, may follow the closure of the fistula if an intra- infection has already been present.

fistula may be closed in the following ways: (1) suture of the dural opening; (2) dural closure by scial transplant; (3) closure of the bony opening flap of dura or other soft tissue; (4) sealing of ny opening with wax.

dy reports 11 clinical cases; 8 of the patients ured permanently by surgical intervention; 2 died acranial infection, and in 1 patient the leakage of fluid persisted because the fistula was not noted ration.

a rule, the location of the fistula can be deter- by the site of the causative fracture or the ive defect in the skull; in rare instances, how- it may be impossible to find the opening. One ation is reported in which a fistula was demon- by injection of methylthionine chloride (meth- blue).

ntaneous pneumocephalus and porencephaly may p as a result of a ball valve mechanism but may ear after closure of the fistula.

fistula through the frontal sinus caused by a de- d fracture is treated by elevating the depressed nts of bone, suturing or covering the dural defect fascia and then replacing the fragments of bone. depressed fracture is present, rhinorrhea is best by formation of a unilateral frontal bone flap side of the leakage. If the fistula is not found, lar operation may have to be performed on the side; at any rate, two small osteoplastic flaps are able to a single large bilateral exposure.

brospinal rhinorrhea does not always indicate he fistula originates from the ethmoid or the sinus; it may develop from an opening in the d bone which drains through the middle ear and stachian tube.

LIST, Ann Arbor, Mich.
[ARCH. NEUROL. & PSYCHIAT.]

THE USE OF PENICILLIN IN OTORHINOLOGIC INFECTIONS: REPORT OF FIVE CASES WITH RECOVERY. SAMUEL D. GREENFIELD, ISADORE KURITZKY and EZRA A. JACOBS, Laryngoscope **55**:20 (Jan.) 1945.

The authors present 5 cases of otorhinologic infection of a type that usually would either be fatal or require operation in which penicillin helped greatly.

The first was a case of thrombosis of a cavernous sinus. Heparin was used in addition to penicillin. Complete recovery ensued.

The second was a case of osteomyelitis of the skull and epidural abscess. The frontal sinus was opened externally and pus was observed. Sulfonamide compounds in large doses failed to prevent the spread of infection. Subsequently osteomyelitis of the frontal bone and an epidural abscess were found. The diseased bone was removed and the abscess drained. Penicillin therapy was instituted at this time, and rapid recovery ensued.

The patients in cases 3 and 4 had mastoiditis, acute in one and recurrent in the other, both of whom, it appeared from the findings, would need operation. Both recovered with penicillin therapy and were still well several weeks later. It is to be noted that the authors strongly advised operation in the case of acute mastoiditis, but permission was refused.

Case 5 was that of a subperiosteal abscess in an infant. There was swelling over the mastoid process and the zygoma and in front of the auricle together with other evidence of a subperiosteal abscess. After myringotomy and penicillin therapy the child recovered quickly. No other operative procedures were needed.

HITSCHLER, Philadelphia.

RADIATION THERAPY OF LYMPHOID TISSUE IN THE NASOPHARYNX AND PHARYNX. HOWARD HICKS ASHBURY, Radiology **43**:250 (Sept.) 1944.

Ashbury reports the results he obtained by treating lymphoid tissue in the nasopharynx and the pharynx with high voltage roentgen radiation. He noted marked improvement in cases of conduction deafness, obstruction of the ostiums and infection when the conditions were due to lymphoid tissue. Five pairs of treatments were given over a period of thirty days. The less responsive patients were ultimately treated by applying radium directly to the lymphoid tissue (75 mg. per hour). One hundred and fifty-seven patients, most of them in the first decade of life, were treated.

ANSPACH, Chicago. [AM. J. DIS. CHILD.]

ROENTGEN TREATMENT OF LYMPHOID TISSUE IN THE NASOPHARYNX. SYDNEY J. HAWLEY, Radiology **43**: 254 (Sept.) 1944.

The object of the procedure here described was to deliver between 700 and 800 roentgens on each lateral wall of the nasopharynx within eight days. The lymphoid tissue disappeared in every instance, and in only 2 of 26 patients was there a recurrence. A second course was given to the 2 patients concerned after six months and resulted in permanent relief. In 17 of 18 patients with impairment of hearing in the author's group there was improvement as measured by the audiometer. Failure resulted when only 600 r was employed.

ANSPACH, Chicago.
[AM. J. DIS. CHILD.]

HYPERTROPHY OF THE THYMUS. D. LONGPRÉ, Union méd. du Canada **72**:901 (Aug.) 1943.

Pediatric opinion is still divided as to the meaning of thymic hypertrophy. The function of the thymus is unknown; hypertrophy may be due to hyperfunction, or perhaps to hypofunction. There is no proof of any thymicolymphatic system. Roentgenographic diagnosis of hypertrophy of the thymus is difficult, and clinical diagnosis is unreliable.

Longpré presents 6 cases in which the clinical diagnosis of thymic hypertrophy was made. In only 1 of these was the diagnosis verified at autopsy, and in case the roentgenologic diagnosis was not definite. 2 cases the condition was probably tetany, but the nosis was not verified. In 2 other cases there congenital lesions of the heart. In the remaining the patient died of massive influenzal pneumonia.

Longpré cites Brennemann in conclusion: "It be a safe procedure to consider the possibility of pressure in all cases of asphyxia, but to realize is an infrequent cause of this symptom."

DAFFINEE, Melrose, Mass. [AM. J. DIS. CH

Book Reviews

Cancer of the Tongue. By Dr. Juan Carlos Oreggia, chief of the Otorhinolaryngologic Service of the Institute of Radiology, assistant to the Chair of Otolaryngology of Montevideo, chief of service of Pedro Visca Hospital, and otolaryngologist to the Institute of Traumatology. Prologue by Prof. Eliseo V. Segura. Cloth. Pages 449, with 145 illustrations and photographs. Montevideo: A. Barreiro & Ramos, 1944.

This excellent book presents a detailed survey of present knowledge of cancer of the tongue. The deductions of the author are primarily based on a series of 158 cases which have been observed within the past fifteen years at the Institute of Radiology. The chapter on etiology is comprehensive and includes all present day concepts on this subject. Chapter 3, which deals with the normal and the surgical anatomy of the tongue and the cervical ganglions, presents the subject in great detail but perhaps could be amplified by a few additional photographs. Chapter 4 deals with pathologic anatomy. This chapter is excellent, describing the various types of cancer and illustrating them with numerous photomicrographs and with case histories from the author's collection. The symptomatology of the condition is presented in an excellent fashion and subdivided to correspond to various periods: (1) the precancerous period, (2) the period in which the lesion is definitely cancer but localized as an indu ulcerative swelling, (3) the period of lymphatic tions, characterized by cervical adenopathy and the period of diffuse infiltration with its m complications. The chapter is amplified by p graphs. Chapter 6 deals with diagnosis, and the subject is given in great detail. Treatme discussed in a clear and concise manner, with p evaluation of surgical procedures and of roentgen radium therapy, with the indication for the use of or both. The author describes (1) partial extirp of the mass, (2) glossectomy of the buccal half o tongue, (3) glossectomy of the pharyngeal part o tongue and (4) total glossectomy. He amply i trates each procedure. He stresses irradiation o involved tissues in its various forms, illustrating various methods of application of both roentgen and radium. A considerable amount of emphasi placed on the methods of radical dissection of the vical glands.

There is a large bibliography, and at the conclu there is a summary of 173 cases with the type lesion, the localization, the complications, the met of treatment and the results.

All in all, the matter is ably and clearly presen and the work is published on good paper with e lent type, easily read.

Society Transactions

ND SOUTH AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY

Montevideo, Uruguay, Nov. 8 to 12, 1944

ORTED BY A. H. PERSKY, M.D., PHILADELPHIA

irst Session, Wednesday, November 8

ylaxis of Hypoacusia. Presented by PROF. V. SEGURA and PROF. E. CASTERAN, of Argentina.

ummarizing their paper, the authors stressed the at the prophylaxis of deafness is of particular t to the physician in general practice, since he e greatest opportunity to take preventive action son of his association with many children as a doctor or with many workers as an industrial ian.

school doctor can (*a*) institute a systematic in- tion of all pupils entering the school, and also se who frequently repeat grades, to discover their g deficiencies, (*b*) institute treatment of the otop- and (*c*) establish special grades and schools e reeducation of those whose hearing is inade-

industrial physician by preemployment otologic nations can detect persons who are susceptible to ational deafness and by periodic examinations of orkers in the industry, by vigilance in controlling orking conditions and by occupational reorienta- of those whose auditory mechanism is deficient e an important preventive mission.

m the standpoint of social medicine the state also bligations to fulfil. By adequate legislation it stablish a better guarantee of careful prophylaxis s grave condition, which signifies a partial or a deficit in the fundamental sense in the everyday the afflicted person.

Postoperative Mastoid Process. Presented by OF. FELIX VEINTEMILLAS, of Bolivia.

e author states (1) that his postoperative results radical mastoidectomy are in general satisfactory t times excellent, particularly in reference to the pearance of the discharge and to cicatrization, if atient realizes that his ear should be maintained the control of a specialist, (2) that they are factory from the standpoint of the local esthetics e area, since there is no development of disfiguring s in persons disposed to these cutaneous neofor- ns, (3) that they are equally satisfactory from tandpoint of good auditory function, provided that ky and irregular epidermization does not form an cle to air conduction, and (4) that the percentage ersons with acute mastoiditis as a complication g those with otitis at the high altitude of Bolivia ecreased 80 to 90 per cent with the administration lfonamide compounds in the last four years.

Second Session, Thursday, November 9

Bronchoscopy as a Means of Diagnosis and Treatment. Presented by DR. PLINIO MATTOS BARRETTO, of Brazil.

In conclusion Dr. Barretto advised that an association be formed of able and competent personnel—administrative, as well as surgical—and that it institute in each of the large hospitals a department of endoscopy, which will easily demonstrate its usefulness and advantages.

This suggestion—which Dr. Barretto wished to have accepted by a vote of every scientific nucleus of importance favoring a center of endoscopy—with opportunities for social prophylaxis and, above all, of incalculable importance, for the treatment of bronchopulmonary diseases—was accepted unanimously by the congress—with a modification proposed by Professor Marinho, who felt that they should ask the Faculty of Medicine of each medical school to establish a chair of endoscopy.

Among other commentators whose remarks Barretto appreciated, Prof. Chevalier L. Jackson said: "I wish to comment in a few words on the official report of Dr. Barretto on bronchoscopy. I wish to say that I am in complete accord with him in what he has said concerning the importance of establishing a center of endoscopy in the large hospital so as to make endoscopy definitely of service, but its importance, as Barretto said, depends on proper locale, on adequate material and particularly on cooperative personnel. Considering these conditions, I believe that it is not important whether or not a bronchoscopist is a laryngologist. As to who should practice bronchoscopy, I feel that this problem should be considered and determined in each city, and in each hospital, taking into consideration the local circumstances and particularly the existing personnel. Bronchoscopy and esophagoscopy have been perfected by the laryngologist, but today one should recognize the right of others to practice it. In some general hospitals it can be practiced by the laryngologist, specially trained in this branch; in sanatoriums for patients with tuberculosis is can be practiced by the phthisiologist or the thoracic surgeon. Furthermore, I believe that today it is justifiable for doctors specializing in anesthesia to practice bronchoscopy, which is so important in the preyention and treatment of postoperative complications.

"In our courses at Temple University we have observed that not only the otolaryngologist but also the phthisiologist, the surgeon, the anesthesiologist and the gastroenterologist are all admitted. We believe that every physician has the right to practice peroral endoscopy, but I am completely in accord with Dr. Barretto that there should be established in each institution a center of endoscopy in the proper locale with adequate material and a cooperative personnel. I am in accord with the suggestion of Dr. Marinho that one should ask the faculties of medicine of the universities to establish chairs of bronchoesophagoscopy, and I believe that in some medical schools these chairs can be chairs of laryngology and bronchoesophagoscopy and in others merely chairs of bronchoesophagoscopy. In the United States the situation is not the same in each locality.

In Philadelphia Dr. Clerf, Dr. Tucker and I practice laryngology and bronchoesophagoscopy. In Chicago Dr. Paul Holinger does, but in other cities the teaching emanates from other sources."

Third Session, Thursday, November 9

The Importance of a Functional Examination of the Eighth Nerve in Cases of Intracranial Tumor. Presented by DR. M. C. RIESCO and DR. C. FERNANDEZ FERNANDEZ, of Chile.

The conclusions of Drs. Riesco and Fernandez Fernandez were as follows:

1. The functional examination of the eighth nerve permits differentiation of lesions of the terminal or peripheral sense organ, the nerve trunk, the central vestibular pathways and the nuclei.

2. This examination, in spite of the insufficient knowledge of the anatomy, the physiology and the pathology of the vestibular system, is a valuable aid in the diagnosis of the localization of certain intracranial lesions.

3. The most important consequence of this failure of exact knowledge of the vestibular system is that the functional examination is only of relative value as a means of examination of spontaneous or induced vestibular signs, of a peripheral or a central type of lesion or of the vestibular syndromes.

4. The value of the results of the functional examination, the relativity of which presents at times a serious obstacle to their correct interpretation, can be supplemented by means of a large personal experience of the otologist and at times by means of his contact with the neurosurgeon and the anatomicopathologist.

5. The relativity of the value of this means of examination exacts otoneurologic experimentation, the personal performance of a complete functional examination and the seeking of symptoms, which must be orientated by a sufficient experience and knowledge of this branch of otology.

6. It is in the presence of a lesion of the posterior fossa that a correct interpretation of the results of the functional examination permits one to make a diagnosis of localization with greater surety. In the presence of a lesion of the anterior or of the middle fossa the results are generally noncontributory.

7. When a lesion at a distance from the eighth nerve or its centers produces a spontaneous or induced cochleovestibular symptom complex so vague and irregular that it permits one to make only a probable diagnosis of the localization, one must think that these findings are due to an action on the internal ear by prolonged intracranial hypertension.

8. By means of the functional examination one can diagnose a lesion at a distance from the eighth nerve, a lesion which directly involves the nerve in its intracranial distribution, a lesion which involves in an irritative manner the nucleus or the central vestibular pathways or a lesion which destroys the nucleus or the central vestibular pathways. Without doubt, experience teaches that the otologist with a large practice should abstain from making a precise diagnosis. It is preferable that, according to the type of syndrome involved, he should suggest a probable relationship between a lesion and the site in which it is possible the nerve is involved.

9. From the previous conclusions one can deduce that the otologist should consider himself to be simply a collaborator of the neurologist or the neurosurgeon whose aid constitutes a clinical element of greater or lesser value to them in their preoperative diag judgment.

10. The efficiency and the value of this collabo are the result of large experience and a permanent tude of investigation.

Fourth Session, Friday, November 10

Rhinoscleroma. Presented by PROF. JUVENAL D GRI, of Peru.

Professor Denegri's conclusions are:

1. Peru is one of the South American co which have foci of rhinoscleroma in their territo

2. In this presentation the statistics are based study of 39 patients coming from diverse localit the country.

3. A search for patients with rhinoscleroma sh be made in every country by trained specialis determine whether or not this disease exists in countries.

4. The diagnosis of rhinoscleroma must be mad cording to the strictest scientific rules, i. e., by hi or by microscopic demonstration and culture o bacillus of Frisch.

5. To combat rhinoscleroma it is absolutely n sary to improve economic conditions, culture in eral and public sanitation.

6. One should establish compulsory registratio patients with this disease in every country in w the disease exists.

7. The treatment of rhinoscleroma should be at expense of the state.

Special Session, Saturday, November 11

Anatomy of the Bronchi. Presented by PROF. C VALIER L. JACKSON, of Philadelphia.

Professor Jackson illustrated his subject with m pictures. The audience this morning was honored the presence of the distinguished phthisiologists P fessors Saya, Sayago and Saenz.

A motion was entertained to render homage to illustrious Prof. Chevalier Jackson, of Philadel the father of the distinguished guest. Professor Se spoke a few words to express the sentiment of entire congress toward this eminent master and condensed his thoughts in the following motion: "T second South American Congress of Otorhinolar gology, completing a special session dedicated endoscopy, in which the work of the South Amer otolaryngologists was considered, profiting by the pr ence of its honored guest, Prof. Chevalier L. Jacks a worthy representative of the famous school of doscopy of Philadelphia, whose founder and cons spirit, Prof. Chevalier Jackson, has made the grea contribution to the perfection of this method of exam tion, which originated in the European schools particularly in that of the illustrious Professor Kil be it resolved to:

"1. Make known to Prof. Chevalier Jackson unanimous vote of this congress their sympathy admiration for that diligent work which has been c pleted in spite of difficulties and pay homage to a totally consecrated to this scientific discipline.

"2. Thank Prof. Chevalier L. Jackson for his pr ence at this congress, to whose brilliant completion has contributed.

Send, by action of the executive committee of the ss, to Prof. Chevalier Jackson a message of tion for the scientific accomplishments of his y son."

geal Cancer. Presented by PROF. JUSTO M. onzo, of Uruguay.

fessor Alonzo reported that in 836 cases of laryn- cancer women were affected in the proportion of r cent and at a later age than men. The lesion ost always an epithelioma, predominantly of the ellular type, and involves especially the glottis. symptom complex has three principal variations: ifficulties of deglutition, later in phonation, with sion high or near the border; (2) glottic dys- , then asphyxia; (3) subglottic asphyxia, then onia (frequently).

diagnosis should establish (1) the existence of and (2) its localization and extent. The second portant for the collaboration of the radiologist in reatment of invasion of the periepiglottic spaces the subglottic region.

treatment the greatest importance is placed on and exact diagnosis. The most difficult and yet ost important procedure is the predetermination equate treatment for each patient. This decision be made without any preconceptions since judg- often presents a margin of error. In general, igher lesions, without differentiation, exuberances ternal appearances, should be treated by roentgen y, and the lower lesions, which infiltrate, im- ize and ulcerate, should be treated surgically.

ally, the treatment should take as a guide the of the life of the patient and, if possible, the of both the vocal and the respiratory function larynx.

ntgen therapy is advanced only to conserve the ion of the larynx, but one must state that the purpose of treatment is to conserve life.

When one does total laryngectomy or extirpation of the epiglotti: and the preepiglottic spaces, one should clean out the carotid ganglion. There have been a definite number of cases in which laryngeal epithelioma metastasized to the ganglion, and life is prolonged after extirpation of the cancer, with or without roentgen therapy.

The author has dedicated his efforts to regulating and improving the operative technic for dealing with cancer of the laryngopharyngeal space, the piriform sinus and the vestibuloepiglottic angle with invasion of the preepiglottic space, where at the best one can do a complete extirpation with conservation of the cords.

There are three types of combined treatment: (1) for the extensive cases, roentgen therapy and, after a period of rest, surgical operation; (2) surgical removal with roentgen therapy; (3) a technic which the author has followed in some cases—one half of the roentgen therapy with operation and then the remaining half of the roentgen therapy.

He tried, with a certain success, the injection of colloidal lead, according to his technic, previous to either roentgen therapy or radium therapy.

An association of syphilis and tuberculosis is present in 10 per cent of the cases and influences the course of treatment.

One should dedicate oneself to the prophylaxis of laryngeal cancer. One should urge avoidance of functional excesses, of irritants, of overindulgence in alcoholic beverages and of syphilis, together with removal of benign but irritating formations, particularly about the age of 35; one should also instruct the public and the physician in general practice how to detect the existence of an early laryngeal stage of epithelioma.

The next meeting of the congress will be called the First Latin-American Congress of Otorhinolaryngology and will be held in San Diego, Chile, within the next three years.

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NATIONAL

All meetings have been canceled.

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Manuscripts for publication, books for reviews and correspondence relating to the editorial management should be sent to Dr. George M. Coates, Chief Editor, 1721 Pine Street, Philadelphia 3, or to any other member of the Editorial Board. Communications regarding subscriptions, reprints, etc., should be addressed, ARCHIVES OF OTOLARYNGOLOGY, American Medical Association, 535 North Dearborn Street, Chicago 10.

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